A PROSPECTIVE STUDY ON THE CORRELATION BETWEEN ADMISSION DAY GLYCEMIC STATUS AND IN-HOSPITAL OUTCOME OF ACUTE CORONARY SYNDROME IN DIABETIC AND NON DIABETIC PATIENTS

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BONAFIDE CERTIFICATE

Certified that the dissertation titled **“A PROSPECTIVE STUDY ON THE CORRELATION BETWEEN ADMISSION DAY GLYCEMIC STATUS AND IN-HOSPITAL OUTCOME OF ACUTE CORONARY SYNDROME IN DIABETIC AND NON DIABETIC PATIENTS”** is a bonafide work of the candidate **Dr. JYOTHIPRIYA JYOTHINDRAKUMAR,** post graduate student, Department of General Medicine, **K.A.P.V.GOVERNMENT MEDICAL COLLEGE TRICHY,** done under my guidance and supervision, in partial fulfillment of regulations of **The Tamilnadu Dr.M.G.R Medical University** for the award of **M.D. Degree Branch I, (General Medicine)** during the academic period from May 2010 to April 2013.

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I solemnly declare that the dissertation titled **“A PROSPECTIVE STUDY ON THE CORRELATION BETWEEN ADMISSION DAY GLYCEMIC STATUS AND IN-HOSPITAL OUTCOME OF ACUTE CORONARY SYNDROME IN DIABETIC AND NON DIABETIC PATIENTS”,** was done by me at K.A.P.V Government Medical College,Trichy under the guidance and supervision of Head of Department & Prof. of Medicine **Prof. P. KANAGARAJ. M.D.** This dissertation is submitted to the **Tamil Nadu Dr. M.G.R. Medical University** in partial fulfillment of the requirement for the award of M.D. Degree in General Medicine.

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CONTENTS

INTRODUCTION

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Coronary artery disease has been considered as the important cause of death in industrialized nations.

Acute coronary syndrome (ACS) includes STEMI and NSTEMI and unstable angina. The important risk factors for ACS are type 2 Diabetes Mellitus (DM), insulin resistance, obesity, cigarette smoking, hypertension, and dyslipidemia.

Unlike other cardiovascular risk factors, obesity and type 2 diabetes are showing a significantly rising pattern.Uncontrolled diabetes has high incidence of ACS and poor prognosis. Higher blood sugar value during admission for ACS carries grave prognosis not only in diabetics, but also in non diabetes patients.

Compared to other risk factors of cardiovascular diseases such as elevated serum lipid levels, cigarette smoking, and hypertension, that are decreasing or better under proper management & control with medicine, obesity & type 2 diabetes are exihibiting increasing trends.(1)

Coronary artery disease which is being considered as the most significant complication of DM, presents two to four folds greater risk of mortality compared to the non-diabetic population.(2) Patient with

diabetes have coronary artery disease much earlier & they show comparatively more wide spread atherosclerosis.(3)

Diabetes Mellitus has been recently considered as an important cardiovascular disease risk factor in the similar group which includes smoking, increased blood pressure and dyslipidemia by American Heart Association.

The Framingham heart study also revealed a marked increased in coronary artery disease, myocardial infarction, congestive heart failure and sudden death in diabetes.

The age matched risk of the above cardiovascular disease is twice in men with DM when compared to those without diabetes.(4)

The Diabetes is showing a rising incidence in the Indian scenario due to ageing of the people and due to high rates of obesity & sedentary living pattern.

Patients with type 2 diabetes who do not have a previous incidence of ACS carry the same risk for coronary artery disease as individuals without diabetes who had suffered a previous episode of ACS.(2) Hence, diabetes should be regarded as an important cause for cardiovascular disease (CVD) & its position should be along with the other known risk factors for CVD. Thus it might be appropriate to say, "Diabetes is a cardiovascular disease. (5)

Out of 4 patients, nearly one patient with ACS has presented with a diabetes history according to Global Registry of Acute Coronary Events. Mostly these individuals belonged to the female gender and they had increased mortality rate; most of them were older age and non smokers. The Framingham Study also confirms that the females had a higher occurrence of diabetes compared to males. (6,4)

In acute MI, DM has been a bad prognostic factor with an increased occurrence of heart failure, higher mortality in the hospital, conduction abnormalities including atrial fibrillation & post-infarction angina among diabetics. (7,8)

It has been shown that adequate control of the blood sugar brings down the progression of microvascular disorder caused by diabetes. However, its influence on macrovascular complications is not well known. (9)

Glucose control may reduce microvascular complications such as nephropathy, retinopathy. This is not so true with cardiovascular adverse effects.

In microvascular complications also, control of hypertension has a better effect than control of blood sugar which independently reduces cardiovascular complications.(10,11)

In uncontrolled cases the condition is grave! According to the Paris Prospective Study, the incidence of death significantly became higher with rising fasting as well as two-hour blood sugar value.(12)

Poor blood control have high incidence of ACS which inturn have poor outcome. Also it is seen that hyperglycemia without history of DM are not uncommon in patients presenting with ACS.(13) Inadequate glycemic management is shown by elevated HblAC, and its elevated value during admission for ACS, increases the mortality in first month. Also hyperglycemia at the time of ACS without the history of DM has increased short term mortality.(14)

Diabetes is a significant risk factor for the development of ACS & the adverse outcome after ACS. ‘Stress hyperglycemia’has been defined in different ways by various studies Transient hyperglycemia has been recognised as a noticeable feature in ACS and is considered to be related to stress (Lakhdar *et al.*, 1984).(15) Sometimes, hyperglycemia can denote pre-existing type 2 diabetes or impaired glucose tolerance which has not been detected before.

Infact increased blood sugar can be an indicator of already prevailing insulin resistance & defective function of beta cell which can result in bad prognosis. Recently hyperglycemia has been related to

increased mortality in diabetic as well as in non diabetic and to an increased incidence of cardiogenic shock (Oswald *et al.*, 1984).(16)

Studies clearly prove that uncontrolled blood sugar has poor outcome when they develop an ACS, but the hyperglycemia at the time of ACS also have similar prognostic value. (17)

We were interested to know what determines the prognosis in a patients admitted for ACS - a persistent high blood sugar level as determined by HbA1c or hyperglycemia noted during the admission.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

CORONARY ARTERY DISEASE

INCIDENCE AND PREVALENCE

Disease of the coronary arteries is the most significant and life-endangering problem that occurs in diabetic individuals. Diabetics have a 2 to 4 times increased risk for CAD compared to non diabetic counterparts.

Cardiovascular disease is the cause for nearly twelve million deaths yearly & is the important reason of death worldwide.

Previously considered as a disease of the affluent, the past three decades have seen considerable decline in the incidence and prevalence of atherosclerotic coronary artery disease in the industrialized western world, whereas at the same time this problem is assuming epidemic proportions in the developing world.

There was a sudden rise in the global prevalence of coronary artery disease in the last century. Global Burden of Disease Study has shown that our nation suffers the highest burden of ischemic heart disease.

Though decrease in deaths due to coronary artery disease is seen in various developed countries, the pattern has been shown to be reversed in developing nations. The above observation is perhaps due to

epidemiological transition happening in those countries. Apparently, the total coronary artery disease deaths from China & India is equivalent to the total deaths due to coronary artery disease happening in all developed nations. A significant ethnic variability is clearly demonstrated in the occurrence of coronary artery disease. It has also been proved that there is an increased incidence of premature ischemic heart disease in Indians.

Asian Indians, whether living in their own country or elsewhere have much higher incidence of CAD as compared to all other ethnic groups.

CAD among Asian Indians have been found to be more severe, diffuse and associated with serious complications and increasing mortality at a younger age.

Coronary artery heart disease related death was an uncommon cause of death in United States at the beginning of the last century, constituting less than ten percentage of total mortality in the year 1910.Mortality level due to CAD rose to 55% of all deaths by the year 1965. The last three decades saw an yearly fall of CAD in the US.

While in India from the 1960's to the 1990's the prevalence of coronary artery disease became elevated to 2 fold (two percentage to four percentage) in people living in rural areas and 3-fold (3.4% to 9.45%) in

Urban populations. The prevalence is even higher in South India (13% urban and 7% rural).

In 1990, 25% deaths in India were attributable to cardiovascular disease compared to 9% due to diarrheal disease, 12% due to respiratory infections and 5% due to tuberculosis.(18)

In India,the pattern of CAD is developing earlier when compared with the age incidence of nations which are considered as developed nations. Incidence in men are greater than females with the time of peak occurrence being observed as from fifty one to sixty years. Elevated blood pressure and blood sugar are the reasons for nearly fourty percent of total cases, and high prevalence of smoking is seen in significant amount of cases.(19)

RISK FACTORS

Atherosclerotic CAD is a fatal disease with no known cure. However, it is preventable and treatable. The National Heart Lung and Blood Institute of USA initiated the Framingham Heart Study in 1949 and by 1961 &the concept of risk factors for CHD was clearly made out.

RISK FACTORS ASSOCIATED WITH THE DEVELOPMENT OF CAD

Non Modifiable Risk Factors	Modifiable risk factors	*Newer risk factors*
Age	Smoking	Atherogenic risk factors
Male gender	Obesity	Lipoprotein(a)
Family History	Diabetes	Elevated Homocysteine level
	High blood pressure	Plasma fibrinogen
	Increased Cholesterol	Tissue plasminogen activator
	Nature of behaviour	Plasminogen activator inhibitor-1
		C-Reactive protein

Hyperlipidemia, elevated serum glucose, increased blood pressure, smoking and family history of premature CAD are proven to be the classically defined risk factors for atherosclerotic coronary artery disease. Newer risk factors include CRP, homocysteinemia and lipoprotein (a).

In ethnic Asian Indians the insulin resistance syndrome(otherwise known as metabolic syndrome X), lipoprotein(a), atherogenic dyslipidemic phenotype and some newer emerging risk factors (homocysteine, tPA PAI-I, fibrinogen , factor VII, infections and inflammation) may be more relevant.The underlying genetic susceptibility associated with a modest abnormality in lipid and lifestyle factors makes CAD assume a malignant course in Asian Indians.(18)

DIABETES MELLITUS

Diabetes is being widely recognised as a major disease of the world. At present it is prevalent in an estimated 143 million individuals globally & its rate is showing a faster growth. About 1-5% Indians have type 2 diabetes are associated complication.

Based on the statistics of last 2 decades, incidence of DM has increased worldwide in drastic proportions, from thirty million in 1985 to one hundred and thirty seven million in 2000. According to the present trend, more than three hundred& sixty million people might be suffering from diabetes by 2030.

Even if both type 1 & type 2 diabetes are having a high occurrence world wide, there is a faster growth in incidence of type 2 DM due to higher obesity rates and decreased activity pattern .This occurs as nations tends to be more industrialized.

This trend appears to be real in most of the countries. In fact out of the top ten countries with higher rates of DM, six are in Asia. It has been estimated that in the United States 20.8 million people (7% of the individuals) suffered from diabetes in 2005, by the centres for disease control and prevention (CDC). In fact nearly 30% of them were undiagnosed.

Diabetes has been listed as the 6th major cause of death in the United States and globally 3 million deaths occur per year.(20)

Criteria for the diagnosis of Type II Diabetes Mellitus (ADA criteria)

Blood glucose in the fasting state greater than or equal to 126 mg/dl (7.0 mmol/l)

OR

Classical Symptoms (such as increased frequency of urination, increased thirst, weight loss which can not be explained) and A random value of blood glucose greater than or equal to 200 mg/dl (11.1 mmol/l)

OR

Blood glucose value greater than or equal to 200 mg/dl (11.1 mmol/l) which is taken two hours after giving a seventy five gram glucose load

OR

HbA1c $\geq$ 6.5%.

DIABETIC BURDEN IN INDIA

In India diabetes has become an important public health issue .It has been reported by WHO that there were 31.7million people with diabetes in our nation in 2000 & this rate might become 71.4 million in

2030. India has turned out to be the diabetic capital with the highest number of diabetics in the world.

A greater incidence of type 2 DM was noticed among Indians who have migrated to other countries for various reasons, when compared with their native population, suggesting that Indians as an ethnic group had a genetic propensity to develop diabetes which was precipitated by life style changes.

Among the Indians living in the urban areas present rate of DM is 12.1%.Datas show that type 2 DM occurs at a younger age amongst Indians, a decade earlier than in the West.

Decline in productivity has occurred as a result of higher rates of morbidity & mortality due to the earlier age of onset, late diagnosis and inadequate care.(21)

DIABETES AND CORONARY ARTERY DISEASE

Diabetics show a greater propensity to develop CAD much earlier than non diabetics and atherosclerotic process in them tends to be more wide spread indicating that the cardiac disease in Diabetic individuals is not similar to that occuring in non diabetics.

According to Multiple Risk Factor Intervention Trial diabetic population with out the conventional risk factors like elevated blood pressure, hypercholesterolemia or smoking had a 4.4 times higher rates of

cardiac deaths when compared with the control subjects of the corresponding age. But patients having HbA1c level less than 6.0% had only a small incidence of CAD.(22)

According to the twenty years of surveillance observed in the Framingham cohort study,it has been reported to have a two-fold to 3 fold greater chance of atherosclerotic disorder prior to evidence of Diabetes. Diabetic females had a greater rates of morbidity & mortality than for non-diabetic males , for each of the CVD.Diabetic females had a similar high CVD mortality rates when compared with the diabetic males.(23)

Diabetic men had a greater risk of CVD death than non-diabetic of all age group, ethnicity, and risk factor level which was shown by a similar study. A 3 times greater rate has been observed after adjustment for age, race, income, systolic BP, serum lipid level and reported quantity of cigarettes per day. A progressive rise in absolute risk of CVD death was observed in diabetic men with higher risk factor than for non diabetic men with similar risk factor levels.(24)

The risk of CVD is substantially higher in diabetic population. Compared to euglycemic individuals, diabetics carry a worse prognosis when clinical cardiovascular disease sets in. Hyperglycemia, by causing defects in endothelium, contributes to atherosclerotic pathogenesis directly.

Elevated blood glucose is a predominant risk factor for formation of cholesterol plaques and subsequent atherogenic mechanism, hyperlipidemia & it is frequently related with high blood pressure, dysfunction of endothelial cells, defective adhesion & aggregation of platelets, coagulation defects and greater oxidative stress.

Atherogenic dyslipidemias tend to be seen in type 2 diabetes. Metabolic syndrome which was previously called as syndrome X includes hyperlipidemia, raised blood pressure & a procoagulant condition apart from the abnormalities in the metabolism of blood sugar.Generally type 2 diabetes or the insulin resistance disorder is seen as a part of metabolic syndrome. Apart from smoking, almost the entire group of risk factors of cardiovascular disease seem to be very much common in type 2 diabetes individuals.

INSULIN RESISTANCE

Diabetic population have an enhanced potential to develop CAD. However, this could not be totally substantiated by the increase of conventional risk factors such as hypertension & hyperlipidemia. But in diabetics the above mentioned factors are proven to be enhanced. A condition of persistently elevated insulin & blood sugar level (hyperinsulinemia & hyperglycemia) has occurred because of the insulin resistance & the subsequent rise in the production of insulin as a part of

compensatory mechanism. Hence its appropriate to say insulin resistance acts as a predecessor of development of DM.

Reaven (25)analysed the relationship of insulin resistance & CAD and he pointed out that DM is seen as a component of the insulin resistance syndrome (known as metabolic syndrome), that comprises elevated blood sugar, disorders of lipid metabolism and truncal obesity. Based on the European Group of Insulin Resistance criteria, Insulin Resistance Syndrome has been described and accordingly, a prevalence rate of 11.2 percentage has been found in South Indians.

MECHANISM OF ATHEROSCLEROTIC PROCESS IN DIABETES

Relationship between high blood sugar, abnormalities of the endothelium and atherogenic process is well understood. In diabetes, the initial prominent atherogenic change is endothelial dysfunction. It includes deranged endothelium mediated vasodilation, which might lead to decreased blood supply& its subsequent consequences by preventing dilatation as well as proliferation of smooth-muscles of blood vessels,enhanced thrombogenesis and proatherogenic cellular processes.

In type 2 DM,there is an established association between the raised plasma glucose level & atherosclerosis. Almost all proteins undergo glycosylation as a result of elevated glucose level. This is causing cross

linkages of collagen and various extra cellular matrix proteins which are seen in the walls of arteries. Prolonged exposure to increased plasma sugar values itself will result in defective function of endothelium. Everything including elevated blood pressure & cholesterol value, enhanced atherosclerotic process and thrombus formation, contribute to the underlying pathophysiology of vascular problems in diabetes individuals. Perhaps it might be the basic defects in endothelial function which is responsible for all these disorders of vascular system.

DIABETIC CONTROL AND CARDIOVASCULAR EVENTS

In patients having established DM, whether adequate blood sugar control alone decreases CVD is an unanswered puzzle.

The diabetic control and complications trial (DCCT) failed to establish a notable decrease in cardiac diseases with adequate control of glycemic status in young subjects with type1 DM. However epidemiology of diabetes interventions and complications (EDIC) trial,which was considered as a follow up study ,succeeded in establishing a delayed benefit.(26)

In various trials it has been observed that adequate glycemic management in subjects with type 2 DM decreases the acceleration of microvascular disorder. However, its impact on macrovascular abnormalities is not clear (27)

Two recent studies that include Action in Diabetes and Vascular Disease (ADVANCE) trial(28) and the Action to Control Cardiovascular Risk in Diabetes (ACCORD) trial(29) failed to observe any significant decrease in cardiac incidents with adequate glycemic management.

Baseline values of HBA1C were 7.2 percentage in ADVANCE study and 8.1 percentage in the ACCORD study. After intensive therapy, glycated hemoglobin levels were reached 6.4% in both studies.

Following adequate treatment strategy, the values became 7.5 percentage & 7.0% respectively. But these trials failed to establish a significant reduction in cardiac incidents. Greater rate of hypoglycemia and increase in body weight was noticed in subjects who were under intensive control. There was not much impact on microvascular consequences by intensive blood sugar monitoring over a duration of five to 6 years.

Adequate blood sugar management, if started in the initial phase of disease, might bring out advantageous effect. But it is essential to avoid severe hypoglycemia. Hence proper control of blood pressure, elevated lipid level and other cardiovascular risk factors can be considered as the efficient strategy for the prevention of CVD among the diabetes.(30)

ACUTE CORONARY SYNDROME

By definition ACS comprises a spectrum of cardiac events which consist of STEMI,NSTEMI & unstable angina.It is diagnosed with the help of ECG,proper history that includes the clinical features and symptoms of ischemia and with the analysis of cardiac biomarkers. The usual ECG changes include T-wave tenting or inversion, ST-segment elevation or depression & pathologic Q waves.

INCIDENCE OF ACS IN DIABETES MELLITUS

Haffner(2) studied seven year incidence rate of ACS in patients with diabetes and prior history of myocardial infarction. The 7 year incidence levels of ACS among non-diabetic population with & without previous episode of MI at baseline has been observed as 18.8% and 3.5 %, respectively (P<0.001). The 7 year incidence levels of ACS in diabetic groups with & without previous episode of MI were 45% & 20.2%, respectively (P<O.001). Thus the myocardial infarction in diabetic subjects is similar to a non diabetic with prior myocardial infarction.

In a study conducted in Iranian population(31) ,the incidence of acute MI has been reported to be higher in diabetic individuals (36.4%) than in those individuals without diabetes(19.2%, p value less than 0.005).

Diabetic subjects with Ml had **greater Blood sugar** (274.7 ± 130.8 vs. 216 ± 89.6 mg/dl, p value less than 0.005), **Fasting glucose level** (222 ± 109.3 vs.167.3 ± 75.6 mg/dl, p value less than 0.01) & **HBA1C** (10 ± 2.4 vs. 9.1 ± 3%, p value less than 0.05) than diabetics having unstable angina. They showed that diabetics having inadequate glycemic management which was proved by elevated glycated haemoglobin value & /or increased plasma glucose & fasting sugar value had greater incidence of Ml.

GENDER DIFFERENCE IN PROGNOSIS OF ACS IN DM

Galcera-Tomas(32) studied gender difference in prognosis of acute myocardial infarction in diabetes. They showed that females with DM suffered a greater incidence of cardiac complications during hospitalization when compared to males with DM. Early incidence of cardiac failure (in diabetic female vs males with DM) was 11.7 percentage versus 4.5 percentage (resulting in a P value less than 0.01), in-hospital incidence rate being 29.6 percentage versus 10.3 percentage (giving a p value of 0.001) & one year rate was 42.7 percentage versus 21.1 percentage (resulting in a P value which is greater than 0.001).

COMPLICATIONS OF ACUTE CORONARY SYNDROME

Complications of ACS include conduction disturbances, mechanical complications, cardiogenic shock and several other complications.

- Arrhythmias :

  1. Ventricular premature beat
  2. Ventricular tachycardia/fibrillation
  3. Accelerated idioventricular rhythm
  4. Supraventricular arrhythmias
  5. Sinus bradycardia
  6. Atrioventricular conduction disturbances
  7. Asystole

- Mechanical complications including free wall rupture, VSD, MR
- Hypotension, Left ventricular Failure, Acute Pulmonary oedema, Cardiogenic shock
- Recurrent Myocardial Infarction
- Pericarditis, pericardial effusion
- Cerebrovascular Accident and thromboembolism
- Left ventricular aneurysm
- Dressler's Syndrome.

MORTALITY OF ACS IN DIABETES MELLITUS

Myocardial infarction in type 2 DM has poor prognosis. There is a greater occurrence of heart failure, in-hospital adverse effects, atrial fibrillation, conduction abnormalities & post-infarction angina. The higher mortality among diabetics is related to an increased occurrence of left ventricular failure.(33)

Lorente (34) analyzed two hundred & thrty five individuals (of which 51.06 percentage were men) with ACS ,DM was found in 26.38 percentage.Individuals with DM had greater incidence of LVF (incidence being 67.74 percentage versus 53.17 percentage) & cardiogenic shock (incidence being 46.77 percentage versus 32.36percentage)

Herlitz(35) studied the mortality, adverse effects & consequences of MI, death pattern & the occurrence of recurrent MI for a duration of five years following ACS in individuals with & without DM. Individuals with DM suffered a 5-year mortality of 72% ,when it was 50% for those with out DM (resulting P value is less than 0.001). Among diabetic patients, 55% developed a re-infarction versus 22% in non-diabetic patients ($p < 0.001$).

Paris Prospective Study(12) followed 7018 men aged 44-55years up to 23 years & found that fasting as well as post prandial levels of insulin have an association with all-cause mortality & the trend showing a

U shaped pattern. The study also proved that those subjects with decreased as well as increased levels of insulin have enhanced incidence of ACS , independently of other risk parameters.It was observed that those individuals having increased levels of insulin had a greater propensity for developing CAD.

HbAlC or GLYCATED HAEMOGLOBIN LEVELS

Glycated or **glycosylated haemoglobin** (*HbA1c*) which is quantified mainly to find out the average plasma glucose level in the past 2 to 3 months.It gives an idea about the chronic glucose control of the subject.The mechanism of nonenzymatic glycation of Hb is involved in its formation.It is formed in a non-enzymatic glycation mechanism by Hb being exposed to plasma sugar.

When the value of blood glucose is with in the reference range,the level of HBA1C also tends to be within the normal limits. With the rise in the mean level of blood glucose, the quantitiy of HBA1C also tends to rise simultaneously. Hence it function as a marker for average plasma sugar values during the past two to three months before the investigation.

HBA1C $\geq$ 48 mmol/mol ($\geq$6.5%) is added in the criteria which has been followed to make a diagnosis of DM.

Elevated levels of HBA1C, suggesting inadequate plasma sugar control, is related with development of various cardiac disease.

UNDERLYING PRINCIPLE

Normally, red blood cells have a lifespan of approximately one hundred and twenty days. People having improperly controlled DM, show a significantly elevated levels of HBA1C when compared to the normal subjects.

When the Hb molecule gets glycated, its a permanent phenomenon. Glycated haemoglobin persists in the same pattern till that red blood cell finishes its life span. Hence the glycated Hb inside RBC, infact, shows the mean value of plasma sugar to which RBC gets exposed in its lifespan. Analysing the value of HBA1C is an indicator of successive treatment through the monitoring of long-term blood sugar control. HbA1c value corresponds to the mean blood sugar level of the body in the prior 4weeks to 3months.But few research workers have the opinion that the greater part of HbA1c assay corresponds to the glycaemic control of the past fourteen to twenty eight days.

MEASURING HBA1C

Several methods have been employed to quantify HbA1c assay.Those methods include:

- High-performance liquid chromatography -In this method glycated haemoglobin value is found out as proportion to complete Hb in the body.

The other methods consist of

- Immunoassay process
- Enzymatic method and
- Capillary Electrophoresis

Diabetes presents with microvascular as well macrovascular pathology. Infact disorders pertaining to macrovascular pathology will be occurring even before a clinical diagnosis of DM has made. Hyperglycemia itself could be considered as a risk factor in the development of CVD. Elevated blood sugar enhances the atherosclerosis process through the production of glycated proteins. There is also production of advanced glycation end products in the body which is a result of elevated blood sugar. They both will result in the worsening of endothelial dysfunction .

Glycosylated Hb can be regarded as a marker of glycated proteins. The various land mark studies have considered the value of HbA1c as an indicator of adequate glucose management. Observations made in Framingham study led to the conclusion that there is a 2fold rise in cardiovascular mortality among males with DM & a 4fold rise in female having diabetes than their corresponding counter-parts without DM.

A profound rise in cardiac adverse incidents and mortality have been noticed when HBA1C level is above 7%. The above mentioned assosciation between raised glycosylated Hb value & elevated cardiovascular adverse events is found itself prior to the establishment of clinical DM .

HbAlC LEVELS AND ACS

In poorly controlled diabetes, as indicated by glycosylated Hb value more than seven percentage, adverse incidents like cardiac failure & reinfaction have been observed.

Substantially greater proportion of individuals having improper glucose management indicated by the glycated Hb value more than seven percentage, showed an higher incidence of Unstable angina, ST EMI, CCF, severely elevated blood pressure, triple vessel diseases & dilated cardiomyopathy .(36)

Findings of UKPDS study pointed that adequate management of blood sugar (indicated by glycated Hb levels < 7%) brought a fall in rate of MI by sixteen percentage. However, association made in this study had not been proved statistically significant.

Mahmut Cakmak (37) studied 100 individuals having ACS and measured blood glucose and glycosylated Hb value within three hours of hospitalization. Subjects have been categorized in to 3sets based on their

glycated Hb value.Those categories consist of glycosylated Hb ranging between 4.5percentage and 6.4percentage(number of individuals were25) 6.5percentage and 8.5percentage (number of individuals were28) & the third set included those values which were greater than 8.5percentage(number of individuals were25. After that entire individuals were subjected to do exercise & imaging studies including the angiography of coronary arteries for the assessment of ischemic scores. A statistically proven association had been made out between glycated haemoglobin value at the time of hospitalization & mortality rates. This association resulted in a p value which is equivalent to 0.009.

Apart from this, a statistically proven assosciation had been also established between elevated glucose value & HBA1C level & complete ischemic scores among the individuals having ACS (resultant P value is equivalent to 0.001).

HYPERGLYCEMIA IN ACS

Elevated blood sugar level in individuals who were suffering from ACS increases the incidence of adverse cardiac events & deaths irrespective of their previous glycemic status.

Stress induced temporary elevation of blood sugar level has been observed in people with out diabetes also.This phenomenon has been

related with several complications which occurs in people with &without diabetes.

PATHOPHYSIOLOGY OF COMPLICATIONS ASSOCIATED WITH STRESS HYPERGLYCEMIA

The pathophysiology of these complications are linked to the occurrence of enhanced oxidative injury, inflammatory process & activation of various enzymes that are known as stress responsive kinases.(38) There is a sudden increase in oxidative stress and exaggerated inflammation due to oscillating hyperglycemia.

Other adverse effects of sudden rise in blood sugar include accelerated rate of apoptosis, abnormalities of endothelial function as well as microcirculation,increased stimulation of coagulation & aggregation of platelets .A positive relationship between hyperglycemia during hospitalization & further complications of ACS had been observed in several studies.(39)

Acute hyperglycemia has an independent correlation between the dysfunction of left ventricle & an extensive infarct area which has resulted from a higher rate of no-reflow mechanism(40,41).Infact research activities conducted in animals proved that sudden hyperglycemia diminishes the ischemic pre-conditioning.(42) It has been also noted that cardiac activity & function have been impaired in

people having elevated blood sugar level along with the development of acute MI.(43)

Acute hyperglycemia leads to a decrease in half-life of fibrinogen apart from causing a rise in levels of fibrinopeptideA, prothrombin, factor VIII, suggesting increased activation of thrombosis.

Thus stress induced sudden elevation of blood sugar level has been observed to be related with inflammation & various other immune reactions. These had resulted in a poor cardiac status & outcome(44).

Research works have proved that an increased cardiac adverse events & related deaths have been found among acute MI individuals having hyperglycemia on hospital admission irrespective of their prior diabetic status.

In a study by Petursson 2006 (45) of the one thousand nine hundred and fifty seven subjects,twenty two percentage gave a diabetic history.

In individuals who were not suffering from DM, hyperglycemia at admission vs normal sugar levels had greater incidence of thirtyday mortality (the incidence being 20.2 percentage versus 3.5 percentage which results in a P value of less than 0.0001) .There was also a greater incidence of late mortality (19.1 percentage versus 11.7 percentage resulting in a P value that is equivalent to 0.007).

The 30-day mortality rate was significantly greater in hyperglycemia at admission without diabetics compared to normoglycemic individuals having diabetes resulting in a P value of 0.002.

ACUTE HYPERGLYCEMIA IN ACS (STRESS HYPERGLYCEMIA)

Hyperglycaemia is often seen in the initial stage of ACS. This transient elevation of blood sugar has been regarded to have association with stress. Individuals having stress related elevation of blood sugar value constitute a separate group having a different course of acute coronary syndrome when compared with those having normal blood glucose level. HbA1c assay serves to differentiate pre-existing DM from that of stress induced elevated blood sugar level.

In patients with acute coronary syndrome, a correlation is drawn between increased blood sugar value at the time of hospitalisation & incidence of complications. In fact this was not dependent on the prior glycemic status. Hence, elevated blood sugar itself would probably lead to adverse events in both DM & non DM subjects.

A controversy has been there whether this elevated blood sugar level is a short term phenomenon or an indicator of underlying diabetic status. At the time of hospitalisation, this sudden rise in plasma glucose concentration could be an indicator of severely damaged myocardium.

However, this stress hyperglycemia is causally associated with subsequent worsening of LV function following reperfusion.

For several years, intolerance of carbohydrates in ACS was subjected to analysis by many researchers. Levine (46) related CAD & DM long back itself (even in 1922 itself) .His observation was CAD independently results in a state of glycosuria which might not have any relation with DM.

Raab and Rabinowitz (47) stated that increased blood sugar value has been a persistent aasosciation of ACS rather than a condition which is related to overt DM. It had been proposed that this might be the result of derangement in the vegetative areas of human cerebrum .This work was carried out in 1936.Ellenberg, Osserman &Pollack (48) suggested a Novel description of elevated blood sugar level in acute coronary syndrome subjects without DM .They suggested that hyperglycaemia could be an indicator of shock. Also it has been considered that increase plasma sugar would be related to enhanced occurrence of abnormalities in conduction as well as rhythm disturbances. This work was carried out in 1952.

Goldberger (49) showed that few individuals with deranged glycemic metabolism at the time of ACS had a tendency to develop DM in future.(This study was in 1962). In 1967,Datey and Nanda (50)

observed that 14 percentage of ACS subjects with dysregulated blood sugar level at the time of acute MI had clinical DM in the subsequent years.

Both Taylor *et al.*, & Allison and Hinton (51)gave a probable mechanism for the dysregulation of glucose metabolism occurring in cardiogenic shocks.The underlying pathophysiology was related to defective insulin production (this work was in 1969). Taylor *et al.*, suggested that production of insulin on IV tolbutamide infusion seems to be supressed in shock that was complicating an acute MI.(52)

Opie, in1971, found that metabolic consequences of ACS have been resembled to those observed in any other condition of acute stress.(53)

Mak *et al.*, have observed that subjects without DM having elevated admission blood glucose value after the first AMI suffered poor immediate outcome. (54) It was shown that the non-diabetics with an elevated admission plasma sugar value have an increased rate of mortality when compared to the diabetics. This might be due to the fact that it is the acute fluctuation of blood glucose that results in accelerated inflammation and increased oxidative stress. Since the blood sugar value of diabetics is already in a higher range, threshold glucose value to cause the various deleterious effects would have been enhanced. The paradoxical resistance

of diabetic heart to ischemic challenge could be yet another probable reason. In a diabetic heart the activity of sodium-proton (Na-H+) exchanger as well as uptake of calcium which carry a significant prominence in reperfusion injury have been decreased. Besides, in DM there is a fall in the glycolytic mechanism which results in decreased accumulation of protons inside the cell. Infact, this could be advantageous to heart particularly in conditions of elevated blood glucose level.

MECHANISM OF STRESS HYPERGLYCEMIA

ACS can be considered as an example of acute stress involving both body & mind. Sudden rise in plasma glucose can be due to increased release of stress hormones, relative decrease of insulin with increased quantity of free fatty acids (FFA).

Parameters resulting in carbohydrate dysregulation tend to be similar to other parameters that increase serum FFA. Hence this demonstrates an intimate association between glucose and FFA metabolism in ACS.

ROLE OF CATECHOLAMINES

Elevation in glycemic value has been the result of increase in stress hormones which include catecholamines & cortisol. Apart from the above mentioned ones, an increase in glucagon & growth hormone have also been invariably noticed.

Due to the activation of human body's sympathetic pathway, elevated catecholamine production occurs. This leads to enhanced glucose formation by liver through various mechanism which includes not only gluconeogenesis, but also glycogenolysis & ultimately resulting in stress hyperglycaemia. Catecholamines have a potential to inhibit the insulin production of pancreatic tissues. Thus resulting in low usage of blood sugar by muscle & adipose cell.

CATECHOLAMINES

In Acute MI, elevated catecholamine secretion happens due to sympathetic stimulation, that can be local or general. The 3 cardiac associated incidents which can result in enhanced sympathetic

stimulation consist of the following

1) Fear &pain of ACS.

2) Hypoxia as well as hypotension which might take place in ACS by activation of neurons in vasculature.

3) Local destruction of cardiac vascular neurons which are present in the infracted heart muscle

GLUCAGON

Glucagon's role in the initial hours of acute MI is not well studied. Hyperglycaemia, per se will not result in raised blood glucagon value in DM unless insulin deficiency has been present.

ELEVATED FFA LEVELS

Besides glucose dysregulation, elevated catecholamine action results in various metabolic derangements such as increase in triacyl glycerols, lipolysis & high plasma FFA in ACS. FFA value will be elevated within thirty minutes of ACS.

HbA1c LEVEL VS ACUTE HYPERGLYCIEMA IN ACS

In a study measuring blood sugar as well as glycated haemoglobin level on hospitalization among 521 subjects with MI.(55) Blood sugar has been grouped in to those less than 7.8mmol/l with sample number being 305, those between 7.8 &11.0 with sample number being 138 or those greater than 11.1 mmol/l with sample number being 78. HbA1c has been categorized in to those less than 6.2 percentage with 420 as sample number or those greater than 6.2 percentage with sample number being 101.

Mean follow-up period had been 1.6+/-0.5 years. In ACS patients, mortality by glucose category had been shown to be 9%, 8% & 25%, respectively (p=0.001); mortality by HBA1C category was 10% vs. 17%, respectively with a resultant P value which is equivalent to 0.14.

Multivariate interpretation showed that glucose category had been strongly related with mortality but HbA1c group was not. Thus increased admission blood sugar seems to be more significant when compared to

previous long-term deranged glucose metabolism in predicting mortality of MI subjects.

Thus admission hyperglycemia can be considered as a significant risk factor for mortality in ACS people .No doubt that Diabetic control appears to delay the development of ACS.Its effective control along with the control of the other risk factors like hypertension, dislipedimia, smoking cessation has more benefit. Uncontrolled diabetes, as indicated by high HbA1c levels, has poor outcome in ACS & admission hyperglycemia appears to have still more grave prognosis.

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AIMS AND OBJECTIVES

AIMS AND OBJECTIVES

AIM

To assess the prognosis of patients with ACS comparing admission blood sugar and HbA1c values.

OBJECTIVES

- To find the blood sugar and HbAIc values at the time of admission in ACS patients.
- To compare the blood sugar and HbA1c values in diabetic and non diabetic patients.
- To determine the age, sex difference in the blood sugar and HbA1c values.
- To find the type of ACS, the adverse cardiac events and condition of the patients at discharge.
- To compare blood sugar and HbA1c values with the patients outcome.

MATERIALS AND METHODS

MATERIALS AND METHODS

The study was conducted at the KAPV Government Medical College, Trichy, Tamilnadu. Patients who were admitted in the general wards and intensive coronary care unit of Mahatma Gandhi Memorial Government Hospital, Trichy with ACS were taken as the study sample.

In this study, we recruited 101 patients with acute MI admitted consequently between May 2011 to November 2012 in MGM Government Hospital, Trichy. The study included ACS patients with or without type 2 diabetes mellitus.

The study protocol was approved by institute's ethical committee. Informed consent was taken from both patient and caretakers.

INCLUSION CRITERIA

Acute coronary Syndrome diagnosed in patients presenting with chest pain and or dyspnoea for >30 minutes and not more than 24 hours with ECG changes

They were classified into

1. **STEMI** - ST segment elevation 1mm or more in two or more contiguous leads with reciprocal ST depression in contralateral leads. ST elevation of 1mm in inferior leads and 2 mm in anterior leads is taken as significant.

2. Non-STEMI - ST deviation in the ECG along with elevation of cardiac biomarkers.

3. **Unstable angina-** ST deviation in ECG without elevation of cardiac biomarkers. A diagnosis of Acute coronary syndrome was established based on clinical features, above ECG findings and cardiac enzyme (CPK) value.

EXCLUSION CRITERIA

Other factors that determine the prognosis of the ACS were excluded

1. Age >75years,
2. Pre-existing renal disease
3. Concurrent systemic infections
4. Past history of ACS and cardiac events
5. Past history of Cerebrovascular accidents

The resident trainee, collected history from the patient and responsible caretakers, detailed physical and systemic examination was carried out.

Following clinical evaluation, ECG was taken and blood was sent for cardiac markers, blood sugar, and HblAc levels.Admission day glycemic status was assessed by sending the blood sample for random blood sugar value and HBA1C level at the time of hospital admission.

Based on the admission blood sugar level patients were categorized in to 2 groups with those having RBS < 140mg/dl & those with RBS ≥ 140mg/dl.A value of 140mg/dl has been taken as cut-off value in accordance with American Heart Association(AHA)(46)

Based on the admission HbA1c level patients are grouped in to 2 category with those having HbA1c less than 6.5% & those with value more than or equal to 6.5%. Routine blood investigations including complete haemogram, renal function test, clotting time and bleeding time were done.

Clinically diagnosis of DM was made if the patient is informed of the diagnosis by a physician prior to the admission or if he was on any therapy for diabetes. ECG has been taken as soon as the patient is admitted & also repeated when indicated. ECHO was done on subsequent day or at the time of discharge.

Cardiaovascular assessment was carried out daily or frequently if required as in complicated cases.The diagnosis, ECG, ECHO reports were discussed with the treating cardiologist or the physician. The findings were recorded in the study proforma. Cases were kept under the close follow up till discharge.

Prognosis of patients was assessed by the condition at discharge and or ECHO findings at discharge.

DATA ANALYSIS

Analysis of data done using SPSS 11.

Independent ‘t’ test, chi square test were used for comparison of data

OBSERVATION AND RESULTS

OBSERVATIONS AND RESULTS

Hundred and one (N=101) patients satisfying the inclusion criteria were assessed

DISTRIBUTION OF PATIENTS BASED ON AGE, SEX AND DURATION OF HOSPITAL STAY

Age:	26-74 Yrs,
Mean Age 57.6 yrs, SD =10.1	
Sex:	Male =77, Female =24
Duration of Hospital stay:	2-14 days, mean 8 days

Table 1: DISTRIBUTION OF PATIENTS BASED ON THEIR

Diabetic history

Known Diabetics	53
Durations of diabetes	0-25 Years : Median =3.87 yrs ; SD=5.8
Treated diabetes	Regularly 48 Irregularly 5
Mode of treatment	Oral hypoglycemic agents (OHA) = 43, Inslulin=9
HBA1C level	Ranges = 4.8-10.2 Mean=7.540
Target organ damage:	Retinopathy 2 Peripheral vascular disease 3 Pervious Cardiac Events 0 Acute DM complications 2 Cerebrovascular damage 1

Up to 50% of the patients has known diabetes and majority of them were treated with oral hypoglycemic agents.

The mean HbA1c levels among the Diabetics were 7.540 indicating a poor control prior to ACS

Table 2: Clinical findings in Acute Coronary syndrome

Risk factors for MI	**Hypertension**		**63**
	Dyslipidemia		**78**
	Smoking		**38**
	Alcohol		**33**
	Obesity		**25**
	Life Stress		**14**
	Post menopausal		**21**
	Family history of CAD		**4**
Chest pain duration	Mean=14 hrs		SD=10 Hours
Dyspnoea	6 patients presented with dyspnoea alone		
Heart rate (/min)	40-150	Mean = 85.7	SD=19.2
Systolic BP	150-220	Mean=135.5	SD=28.6
Diastolic BP	0-120	Mean=87.1	SD=19.4

47% of patients had 3 or more risk factors for ACS

Table 3: Investigations

Cardiac enzymes:	**CPK Positive =52**
Renal Functions testes	
Blood urea	12-49
Serum creatinine	0.6-1.5
Haemogram:	
Hb%	9-18
PCV	25-51
Total leukocyte count	4000-15800
Platelets	1.6-5 lakhs
CT	7-10 min
BT	2-4 min

The table shows that the renal functions test, and routine haemogram, and bleeding parameters were in normal limits, the high total leukocyte count is expected in ACS

Table 4: Admission RBS and HbA1c

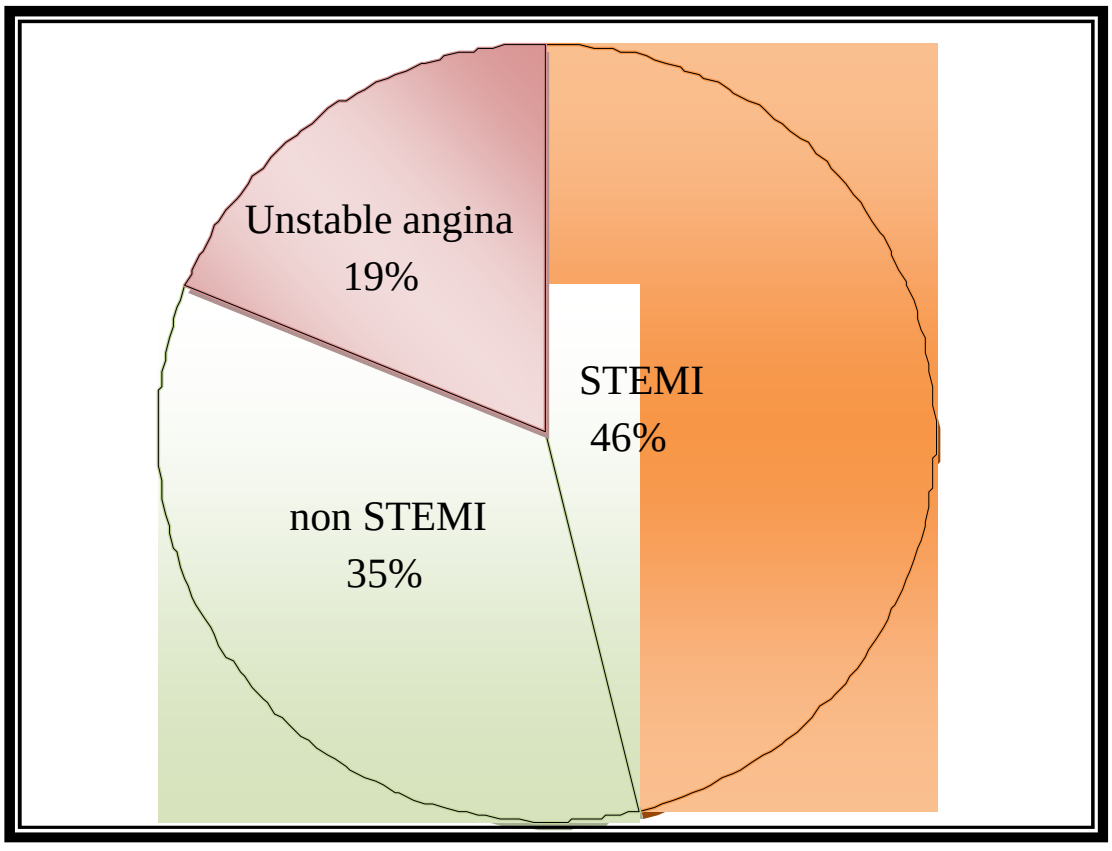
Admission Day Random blood sugar (RBS)	**N=101 Range=66-592mg%** **Mean=220.7 mg% Sd=114.1**
Admission RBS≥140mg%	N=72 Mean = 265.6mg% 44 were known diabetic (mean = 287.1mg%). 28 were non-Diabetic (mean=231.9mg%)
Admission RBS<140 mg%	N=29 Mean=109.3mg% 9 were known diabetic (mean=116mg%) 20 were non diabetic (mean = 106.3mg%)
Admission HbA1c	N=101 Range=4.7-11.0 Mean=6.8, SD=1.51
Admission HbA1c ≥ 6.5	N=59 Mean = 7.89 45 were diabetic (mean=7.89) 14 were non diabetic (mean=7.65)
Admission HbA1c < 6.5	N=42 Mean = 5.5 8 were known diabetic (mean=5.57) 34 were non diabetic (mean=5.48)

Table 4A: RBS, HbA1c in Diabetic and Non Diabetic patients

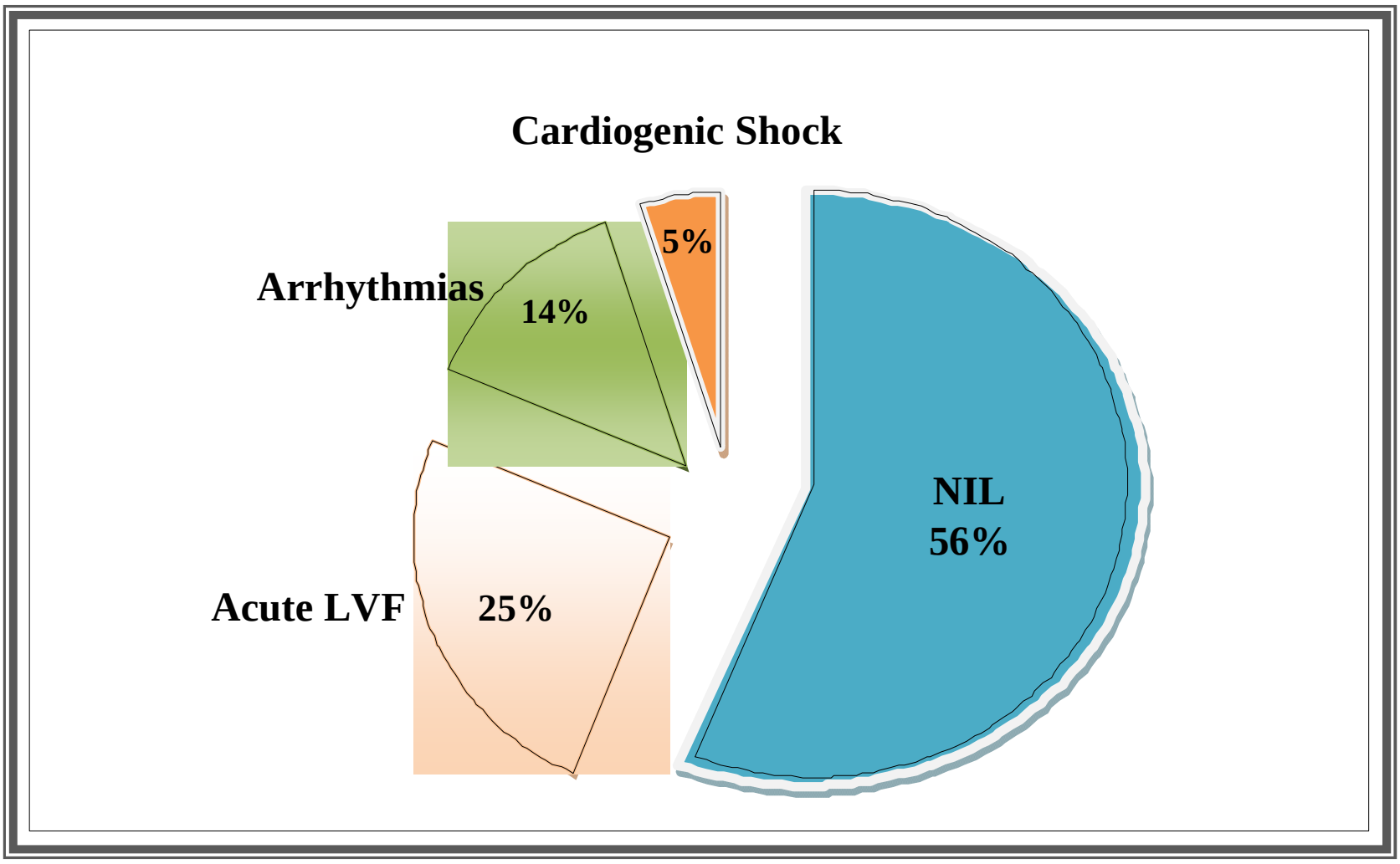
		N	Mean	Std Deviation	Std. Error Mean	P value
RBS	Non DM DM	48 53	178.49 260.65	93.506 118.226	13.358 16.395	.0001 .0001
HbA1c	Non DM DM	48 53	6.151 7.540	1.3523 1.3590	.1932 .1885	.0001 .0001

The blood sugar among the diabetics had high admission RBS and HbA1c values when compared to the non-diabetics presenting with Acute coronary syndrome

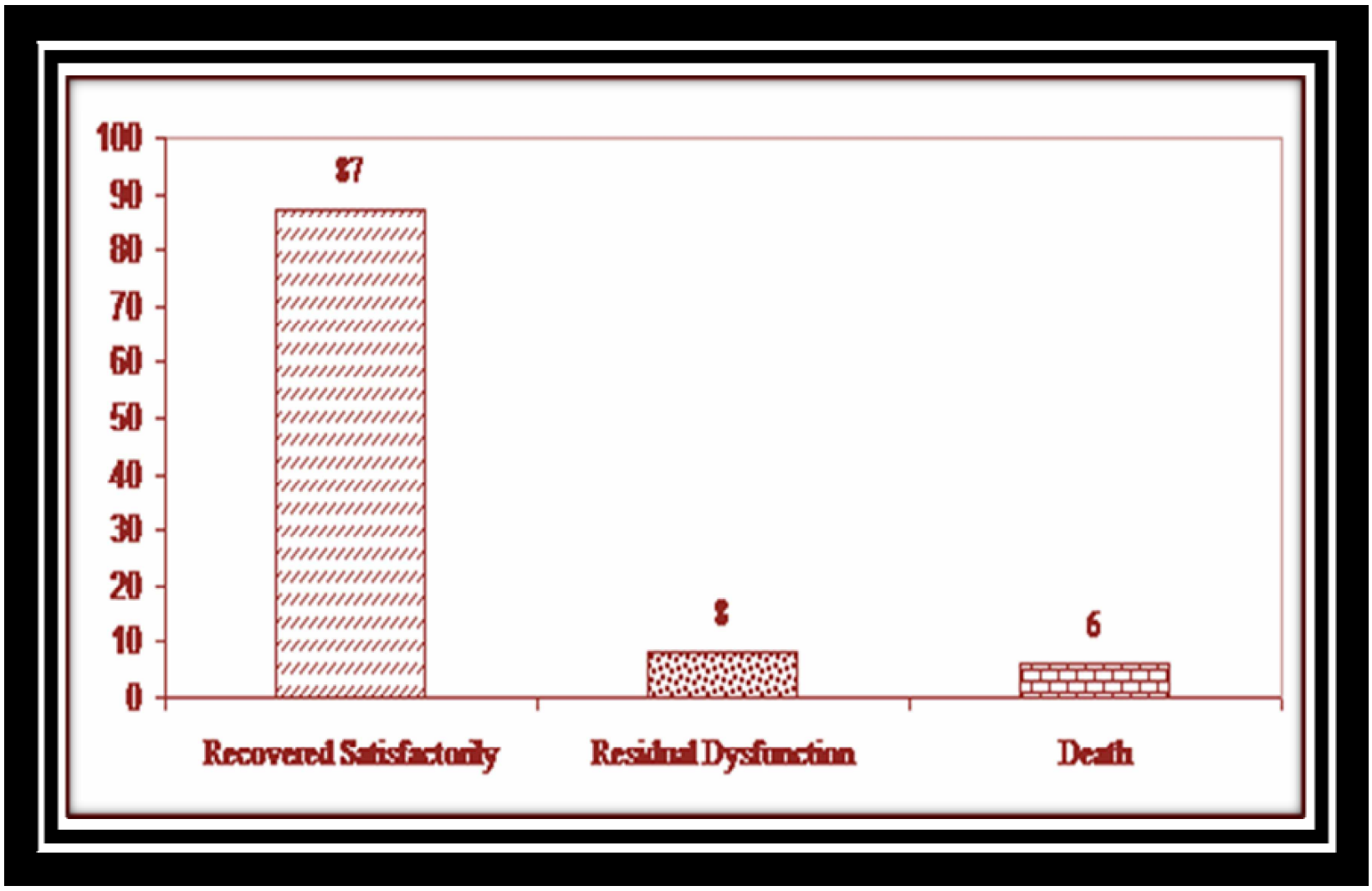
DiagnosIs



Adverse Cardiac Events (ACE)



CARDIAC OUTCOME



Acute left ventricular failure,

Cardiogenic shock and

Ventricular arrhythmias.

Table 5: Admission blood sugar, HbA1c and Condition at discharge

		N	Mean	Std Deviation	Std. Error Mean	P value
RBS	Recovered Poor outcome	87 14	219.29 230.14	119.442 75.708	12.806 20.234	.743 .654
HbA1c	Recovered Poor outcome	87 14	6.844 7.007	1.532 1.475	.1643 .3943	.711 .706

87 had recovered satisfactorily, 14 had residual dysfunction or death. There was no statistical difference in admission RBS or HbA1c with respect to condition at discharge.

Table 6: Admission blood sugar, HbA1c and Adverse cardiac Events (ACE)

		N	Mean	Std Deviation	Std. Error Mean	P value
RBS	**ACE absent**	**57**	**179.82**	**82.52**	**10.93**	**.0001**
	ACE present	**44**	**273.86**	**127.76**	**19.26**	**.0001**
HbA1c	ACE absent	57	6.747	1.59	0.21	.37
	ACE present	44	7.020	1.42	0.21	.36

Adverse cardiac events (ACE) were found in 44 patients

Admission RBS were significantly high in patients with adverse cardiac events while HbA1c was not

Table 7 A: Diabetic patients and Adverse cardiac events with ADMISSION RBS VALUE

RBS	ACE Present	ACE absent	Total
<140mg %	2	7	9
≥140mg %	21	23	44
Total	23	30	53

Chi square test ($\chi 2$) = 3.066 Degree of freedom (df)=1

P>0.05 Null hypothesis is true

Table 7B: diabetic patients and adverse cardiac events with ADMISSION HbA1c VALUE

	ACE Present	ACE absent	Total
HbA1c<6.5	3	5	8
HbA1c≥6.5	20	25	45
Total	23	30	53

Chi square test ($\chi 2$)=0.1335 Degree of freedom (df)=1

p>0.5 Null hypothesis is true

Table 7C: Non-Diabetic patients and Adverse cardiac events

	ACE Present	ACE absent	Total
<140mg%	4	16	20
≥140mg	17	11	28
Total	21	27	48

Chi square test ($\chi 2$) = 7.84 Degree of freedom (df)=1
P > 0.005 Null hypothesis is true

Table 7D: Non-Diabetic patients and Adverse cardiac events with HbA1c

	ACE Present	ACE absent	Total
HbA1c<6.5	14	20	34
HbA1c≥6.5	7	7	14
Total	21	27	48

Chi square test ($\chi 2$) = 0.313 Degree of freedom (df)=1

p>0.5 Null hypothesis is true

The above four tables (7A, 7B, 7C, 7D) shows that patients with no history of diabetic when presented with admission RBS ≥ 140mg% has significant adverse cardiac events. While HbA1c values did not predict adverse cardiac events both in diabetic and diabetic patients

Table 8: Mean age difference among DM & non DM

			Mean age (years)	*Std Deviation*	*Std. Error Mean*	*P.Value*
Age	Non DM	48	56.08	12.261	1.752	.141
	DM	53	59.08	7.590	1.052	.147

Table 8A: Age of patients AND cardiac out come

		N	Age mean (Yrs)	Std Deviation	Std. Error mean	P.value
Age	ACE absent	57	56.25	10.029	1.328	.122
	ACE present	44	59.41	10.228	1.542	.124
	Recovered	87	57.56	10.438	1.119	.883
	Poor outcome	14	85.00	8.840	2.363	.869

There were no statistical difference between the age of Diabetic patients compared to non diabetic patients.

The patient’s age was not statistically significant with the adverse cardiac outcome (ACE) or the condition of discharge.

Table 9: Gender difference IN admission RBS and HbA1c VALUES

		N	Mean	Std Deviation	Std. Error mean	P value
RBS	Male	77	218.27	110.95	12.64	.693
	Female	24	228.88	126.01	25.72	.714
HbA1c	Male	77	6.88	1.476	.168	.844
	Female	24	6.81	1.678	.342	.854

There was no gender difference in admission RBS and HBA1C values in patients presenting with ACS

Table 9A: Gender difference and Adverse Cardiac Events (ACE)

	ACE Present	ACE absent	Total
Male	32	45	77
Female	12	12	24
Total	44	57	101

Chi square test (X2) = 0.515 Degree of freedom (df) =1

P>0.05 Null hypothesis is true

There were no gender difference with respect to adverse cardiac events.

Table 10A: duration of Diabetes and ACE

		N	Mean (years)	Std Deviation	Std. Error Mean	P Value
Duration of DM	ACE absent	57	3.29	5.260	.697	.262
	ACE present	44	4.61	6.535	.985	.276

Duration of diabetes were unrelated to the adverse cardiac outcome

Table 10B: Duration of hospitalization and Adverse Cardiac Events (ACE)

Duration of DM		**N**	**Mean**	**Std Deviation**	**Std. Error Mean**	**P Value**
	ACE absent	57	7.95	1.216	.161	.001
	ACE present	44	9.23	2.381	.359	.002

Patients with adverse cardiac events were hospitalized for longer duration

DISCUSSION

DISCUSSION

THE STUDY SAMPLE:

Our study population included only patients admitted with ACS with or without history of type 2 Diabetes.

Comorbidities such as renal disease, Cerebrovascular accident, previous history of Ml concurrent infections were excluded, so as to study the prognosis related to the diabetes and blood sugar alone.

DIABETIC HISTORY

Diabetes comprised just above 50% of the sample (N=53) and majority of them (81 %) were treated with oral hypoglycemic agents. Complications related to Diabetes comprised less than 5%.Mean HbA1c among diabetics was 7.54% indicating a poor control prior to diabetes. This high value of HbA1c also indicates that though majority of the diabetics have received some treatment with either oral hypoglycemic agents or insulin, the treatment was not adequate. This may be either due to lack of compliance or improper follow up.

CLINICAL FINDINGS IN ACS

Apart from diabetes upto 47% of the patients had 3 or more risk factors for ACS, majority of them being hypertension, dyslipidemia, and smoking. Other risk factors observed in our study include obesity, life

stress, post menopausal state and family history of diabetes. Similar observations were noted in several other studies which have proven that hypertension and dislipidemia are more frequent comorbidities in diabetics.(3)

THE BLOOD SUGAR VALUES

ADMISSION RBS AND HbA1c

Random blood sugar values measured in our study population fell in the range of 66mg% to 592mg%.In our study,72 patients had admission RBS ≥ 140mg/dl. Among this 44 patients were known diabetics.That means 71% of the study population presented with acute hyperglycemia irrespective of their diabetic status. And among the hyperglycemia group 61% were having previous history of diabetes. Only nearly 29% patients presented with an admission RBS value <140mg/dl which clearly shows that in patients with ACS there is a high incidence of elevated admission plasma glucose value.

In this study, 59 patients had admission HbA1c value ≥6.5, of which majority, ie 45 patients (more than 76%) were known case of diabetes. Fourteen patients without a previous history of diabetes showed an elevated admission HbA1c value. These patients might be suffering from diabetes as indicated by an elevated glycated Hb value which was undiagnosed so far.

ADMISSION RBS & HbA1c IN DIABETIC AND NON DIABETIC POPULATION

The mean blood sugar among the diabetes was 260.65 mg% and non diabetes 178.49 mg%

The mean HbA1c among the diabetes was **7.54** and non diabetes **6.15,** the difference was statistically significant.From these it is evident that the diabetic population had both high admission RBS and poor control prior to ACS.

This is in concurrence with earlier study by Esteghamati(31) found myocardial infarction was more frequent in diabetic patients compared to non diabetic patients.

Diabetic patients with Ml had significantly higher blood sugar, and HBA1C compared to non diabetics. Also it has been found that the occurrence of CAD tends to be decreased in subjects having HbA1c level less than 6.5 %.(22). Studies have shown that individuals having higher HbA1c value have very high risk of CVD. (56,57)

ACS AND CLINICAL OUTCOME

Our study showed that 46% had ST elevation Ml, 35% non ST elevation Ml and 19% had unstable angina. While population based studies have shown that up to 23.1% of patients presented with ACS has ST elevation Ml.

The common causes of mortality in the diabetics were heart failure, atrial fibrillation, conduction abnormalities and post infarction angina.

Diabetic patients shows larger infarct size, increased occurrence of left ventricular failure. (33)

In our patients acute LVF, arrhythmias, cardiogenic shock were found in 25,14,5 % respectively of which 6 patients died mainly due to Left ventricular failure, ventricular arrhythmias and cardiogenic shock.

In this study, the most common adverse cardiac event observed was left ventricular failure. Several other studies have also made the similar observation. Cardiac failure had been very frequently associated with increased admission plasma sugar value in MI patients. (58).

ADMISSION BLOOD SUGAR, HbA1c AND CLINICAL OUTCOME

During discharge from hospital 87 had recovered satisfactorily, 8 had complications such as residual ventricular dysfunction, AV block, ventricular septal rupture, mitral regurgitation and 6 patients had died.

There was no statistical difference in mean values of admission RBS and HBA1C with respect to the outcome of the patient during discharge, probably due to the intervention during the hospital stay.

In our study the adverse cardiac events such as acute LVF, Ventricular arrhythmias, cardiogenic shock were found in 44 patients

following ACS; they had longer duration of stay in hospital, and this was unrelated to the age and sex of the patients, or duration of diabetes.

The patients with adverse cardiac events had a mean admission RBS& HbA1c of 273.86mg% and 7.020 respectively, while it was 179.82mg% and 6.747 for those without adverse cardiac events.

In this study the mean admission RBS were significantly high in patients with adverse cardiac events while mean HbA1c was not.

Moreover patients presenting with above normal admission RBS (≥140mg%), had more adverse cardiac events. This was statistically significant in non diabetic patients.

This study had shown that patients with no history of diabetes when presented with an elevated admission RBS have significant adverse cardiac events when compared to a diabetic patient with elevated admission RBS**.**

Similar observation was made in various other studies. In the study by Mak *et al*, it has been shown that subjects without DM having elevated admission blood glucose value after the first AMI suffered poor immediate outcome. (54)

In our study HbA1c values did not predict adverse cardiac events both in diabetic and non diabetic patients.

Several studies discussed about whether the role of admission glucose levels was more important than diabetes history in patients with AMI. Goyal A, *et al.*,. found people having ACS with admission blood sugar value of more than 144mg/dl had an increased rate of mortality irrespective of prior diabetic status.(59) Admission glucose levels in non-diabetic patients with AMI could also offer a good screening tool to evaluate the high risk for future type II DM patients.(60)

Hsu CW, *et al.*,. also found an increased initial glucose value in the emergency department was an independent predictor of short as well as long-term adverse prognosis in individuals first MI.(61)

Individuals with a prolonged history of DM more often have signs of diabetic neuropathy which can cause atypical symptoms during MI . Hence, diagnosis of MI has become a challenge in such subjects and initiation of proper treatment has often prolonged.

Studies have shown that in individuals having ACS, DM has been related with greater mortality rates, both within the hospital as well as during long-term follow-up. (62) In fact, this tends to be the pattern in the entire ACS category.

Elevated plasma sugar value in subjects hospitalised for MI seems to be a frequent phenomenon. Studies have pointed out that there is a

greater rate of mortality and other complications due to this elevation in both group of individuals with & without DM.(63)

The correlation between enhanced plasma glucose on hospitalization & adverse consequences might be the parameter which is independent of other prognostic determinants. (64)

AGE AND SEX DIFFERENCE BETWEEN DIABETICS & NON DIABETICS

In our study mean age of diabetic ACS patient was 59 years and that of non diabetic was 56 years indicating the absence of a statistically significant difference between age of diabetic patients when compared to non diabetic patients.

There were no age and sex preponderance between the diabetic and non diabetic population in our study indicating it is a homogenous sample.

GENDER DIFFERENCE IN ADMISSION RBS & HbA1c VALUES

Of the total population,77 were males & 24 were females. The mean RBS value and HbA1c values among males were 218.27mg% and 6.88% respectively, and that of females were 222.88mg% and 6.81% respectively. Our study shows that there is no gender difference in admission RBS & HbA1c values in patients presenting with ACS.

GENDER DIFFERENCE & ADVERSE CARDIAC EVENTS(ACE)

In this study, of the total 77 males 32 developed ACE and of the total 24 males 12 developed ACE. The present study showed that there were no gender differences with respect to adverse cardiac events.

But epidemiological studies have shown that women have greater incidence of proven DM than males and a increased relative hazard of death compared to non diabetic ladies.(4)

DURATION OF DIABETES & ACE

Our study showed that duration of diabetes was unrelated to the adverse cardiac outcomes.

DURATION OF HOSPITALIZATION & ACE

This study shows that patients with ACE had a longer duration of stay in hospital.

Admission hyperglycaemia seems to be a powerful predictor of mortality even in subjects without a diabetic history. Hence admission blood sugar value of MI can be taken as an indicator to find out high risk subjects so that proper treatment & careful monitoring could be initiated very early itself.

Both HbA1c and admission glucose may have relation with adverse prognosis.However,our results suggest that increased admission glucose is more important. The less clear association between HbA1 C

and prognosis in our analysis could be due to a limited number of patients with a relatively short follow-up in our study.

Limitations of study:

The study was limited to the hospital stay only, follow up of these patients was not done.

Long term follow up study is required to evaluate the correlation between HbA1c values and outcome of the patients with ACS.

Large sample size is required to confirm the age, and gender difference in ACS outcome.

CONCLUSION

CONCLUSIONS

The following conclusions could be made out from this study

- More than 50% of the patients had known diabetes, indicating that the diabetic patients have a greater incidence of ACS
- Mean HbA1c among diabetics was 7.54% indicating a poor control prior to diabetes. This is inspite of the fact that the majority of them were treated with oral hypoglycaemic agents. This indicates that the current treatment received by the diabetics in our study group is inadequate.
- The Diabetic patients had high admission RBS value and HbA1c values when compared to the non-diabetics presenting with ACS.
- Adverse cardiac events occurred in nearly 44% of ACS patients
- The most common adverse cardiac event is acute left ventricular failure.

Admission RBS were significantly high in patients with adverse cardiac events while HbA1c was not.

- Patients with no history of diabetes when presented with an elevated admission RBS have significant adverse cardiac events
- Even though high HbA1c values in a diabetic population is a predictor of acute coronary syndrome, HbA1c can't be used as a marker to predict immediate adverse cardiac events during an ACS in both diabetics &non-diabetic patients .

- There were no age and sex preponderance between the diabetic and non diabetic population
- There was no gender difference in admission RBS & HbA1c values in patients presenting with ACS.
- There were no gender differences with respect to adverse cardiac events.
- Duration of diabetes was unrelated to the adverse cardiac outcomes.
- Patients with adverse cardiac events had a longer duration of stay in hospital.
- Admission hyperglycaemia seems to be a powerful predictor of mortality even in subjects without a diabetic history.
- Uncontrolled diabetes as indicated by high HbA1c values is a strong predictor for development of Acute coronary syndrome.
- However, acute disturbances in glucose levels as indicated by high admission RBS appears to be of greater importance in predicting short term outcome.
- Hence admission blood sugar value of MI can be taken as an indicator to find out high risk subjects so that proper treatment & careful monitoring could be initiated very early itself.

SUMMARY

SUMMARY

Our study was a prospective study connducted to analyse the assosciation between the admission RBS and HbA1c values with the outcome of ACS patients during the hospitalization period.Study was done in 101 patients which included both diabetics & non-diabetics.

Of the total patients 53 were type 2 diabetes mellitus. Mean blood sugar among diabetics was 260.65mg/dl and non diabetics 178.49 mg/dl and mean HbA1c among diabetics was 7.54 and non diabetics 6.15. The difference was statistically significant indicating the diabetic population had both high RBS and poor control.

46% had ST elevation Ml, 35% non ST elevation Ml and 19 had unstable angina. Adverse cardiac events such as acute left ventricular failure, ventricular arrhythmias and cardiogenic shock were found in 44 patients. During discharge 87 had recovered satisfactorily, 8 had complications such as residual ventricular dysfunction, AV block, ventricular septal rupture, and mitral regurgitation. Six patients had died due to left ventricular failure and ventricular arrhythmias.

There were no age and sex preponderance with adverse cardiac events, and the duration of diabetes, while these patients had longer hospital stay.

The mean admission RBS were significantly high in patients with adverse cardiac events while HbA1c was not.

Also when patients presented with admission RBS ≥140mg% had significantly higher adverse cardiac events especially in non diabetic patients.

There was no statistical significant association between HbA1c and adverse cardiac events in diabetic as well as non diabetic patients.

Both HbA1c and admission glucose may have relation with adverse outcome. However, our results suggest that increased admission glucose is more important. The less clear association between HbA1c and prognosis in our analysis could be due to a limited number of patients with a relatively short follow-up in our study.

RECOMMENDATIONS:

Blood sugar control among diabetics is necessary to prevent ACS.

Blood sugar control in ACS patients is essential to improve its outcome whether the patient had diabetes or not.

Long term follow up study is required to evaluate the effect of uncontrolled diabetes following ACS.

From this study it is clear that higher admission blood sugar value in ACS is a powerful predictor of increased incidence of in-hospital adverse events, especially in subjects without prior history of diabetes.

Also it is evident that hyperglycemia is very frequent in the setting of acute coronary syndrome and mostly this is left untreated.

Hence acute hyperglycemia in ACS needs early detection and prompt management should be carried out inorder to have a better outcome.

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APPENDICES

ABBREVIATIONS USED IN THE DISSERTATION

ACS	-	Acute Coronary Syndrome
AMI	–	Acute Myocardial Infaction
DM	–	Diabetes Mellitus
CAD	–	Coronary Artery Disease
CVD	–	Cardio Vascular Disease
STEMI	–	ST Elevation Myocardial Infaction
NSTEMI	–	Non St Elevation Myocardial Infaction
LVF	–	Left Ventricular Failure
CCF	–	Congestive Cardiac Failure
ACE	–	Adverse Cardiac Events
FFA	–	Free Fatty Acids

PROFORMA

STUDY PROFORMA

Name: IP NO:

Age / Sex DOA

Diabetes DOD

Duration:

Treatment history (treated regularly/Irregularly)

(OHA/Insulion/both)

Any target organ damage: Retinopathy,nephropathy,CVA,peripheral vascular disease

Any other diabetic complications

Comorbid conditions (hypertension,dyslipidemia,obesity,smoking,stress)

Presenting complaints

Positive findings.

Investigations

Admission RBS

Admission HBA1C

ECG

ECHO

Cardiac enzymes

Urea/creatinine

Hb	PCV
TC	platelet
CT	BT

Adverse cardiac events

Condition at discharge

(recovered /residual dysfunctions / death)

Diagnosis

(ACS STEMI, NSTEMI, Unstable angina)

age	sex	dm +/-	dur of hos	dur of DM	treated	type	retinopa	PVD	pre car e	nephro	cva	Dm comp	HT	dyslip	alcoho	obesity	smoking	stress	**Any other**	cp hrs	**dyspnoea**	HR	BP SYS	BPDIA	**CVS**	RS	**PA**	RBS	**HB1AC**	ECG	**ECHO**	CARD ENZY	**UREA**	S Creat	Hb	TC	Plt	PCV
67	1	1	8	10	1	3	0	0	0	0	0	0	1	1	1	0	0	0		6	0	92	180	80	1	1	1	346	10.1	St dep anterior leads	no rwma,n lvf	0	33	1.2	13	7500	4	39.6
46	1	0	7	0	0	0	0	0	0	0	0	0	0	0	1	0	1	0		3	0	84	110	70	1	1	1	203	9.6	st ele inferior leads	hypo rca mild Lvd	0	13	0.8	12.6	10000	2.25	39
49	2	1	9	7	1	1	0	0	0	0	0	0	1	1	0	0	0	0	pt hysterectomy	10	0	90	150	90	1	1	1	297	8.3	st dep anterior leads	hypo Lad n Lvf	0	15	0.8	8.9	10300	2.68	25
55	1	1	7	5	1	1	0	0	0	0	0	0	0	0	1	1	1	0		22	0	63	120	90	1	1	1	231	8.6	st ele anterior leadfs evolved	hypo lad mild Lvd	0	17	1.1	15.4	11200	4.35	44.4
60	1	1	8	5	1	1	0	0	0	0	0	0	1	1	0	0	0	0		3	0	102	190	120	2	2	1	289	9.4	st dep anterolateral leafs	hypo lad severe Lvd	1	24	1.3	17.7	5400	1.2	45
70	1	1	7	1.5	1	1	0	0	0	0	0	0	1	1	0	0	0	1	post menopause	20	0	75	130	100	1	1	1	164	6.5	lbbb	global Lv hypo, severe Lvd	0	17	1.2	13	10400	3	40.6
65	1	1	9	12	1	1	0	0	0	0	0	0	0	0	0	0	0	0		18	0	114	100	70	1	2	1	339	7.2	st ele anterior leads evolved	global Lv hypo, severe Lvd	1	40	1.4	12.9	17200	2.3	37
58	2	1	7	6	2	1	0	0	0	0	0	0	1	1	0	1	0	0	post menopause	24	0	76	120	80	1	1	1	147	4.8	st ele anterior leads evolved	hypo lad mod Lvd	1	18	1.2	9.6	10000	4.5	29
63	1	1	13	3	2	1	0	0	0	0	0	0	0	1	0	0	0	0		20	0	80	136	90	1	1	1	533	8.6	ele anterior leadfs evilved	global Lv hypo, severe Lvd	1	43	1.2	11.7	14200	3.6	34.8
53	2	1	7	10	2	0	0	0	0	0	0	0	1	1	0	1	0	0	post menopause	4	0	82	150	100	1	2	1	233	9.3	str dep anterior leads	global Lv hypo, severe Lvd	1	36	1	11.3	8200	2.7	35.3
74	1	0	12	0	0	0	0	0	0	0	0	0	1	1	0	0	0	0		0	4	96	170	110	2	2	1	473	5.6	str dep anterior leads	hyo Lad mod Lvd	1	33	1.4	13.5	23800	2.9	41.8
52	2	1	7	1	1	1	0	0	0	0	0	0	1	1	0	0	0	0	post menopause	6	0	92	160	110	1	1	1	244	8.6	str dep anterior leads	hyo Lad mod Lvd	1	12	0.9	13	8300	2	40.2
63	1	1	10	18	1	1	0	0	0	0	0	0	0	1	1	0	1	0		5	0	116	120	80	1	1	1	406	5.9	st ele anterior leads	hyo Lad mod Lvd	1	34	1	14.4	11500	3.4	43
50	1	1	2	3	1	1	0	0	0	0	0	0	0	1	0	0	1	0		3	0	100	160	110	2	2	1	219	8	st ele anterior leads	hyo Lad mod Lvd	1	20	1.2	15.3	16800	4.1	44.5
70	1	1	7	10	1	1	0	0	0	0	0	0	1	1	0	0	0	0		4	0	102	170	110	2	2	1	280	5.9	st ele anteior leads evolved	hyo Lad mod Lvd	1	31	1.3	11.5	15000	1.9	38
74	1	0	11	0	0	0	0	0	0	0	0	0	1	0	0	0	0	0	Bph	0	4	80	170	120	2	2	1	473	5.6	st dep lateral leads	hyo Lad mod Lvd	1	33	0.9	13.5	13800	2.4	41
72	1	0	7	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0		9	0	102	210	100	1	2	1	250	6.3	st dep anterior leads	hyo Lad mod Lvd	0	27	1.2	12.6	13100	3.5	38
45	2	0	7	0	0	0	0	0	0	0	0	0	0	0	0	1	0	0	pt hysterectormy	22	0	80	140	90	1	1	1	100	5.3	st depinferior leads	no rwma, n Lvf	0	23	0.9	13.7	9000	4	40
70	1	0	7	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0		3	0	54	100	70	1	1	1	119	5.5	st ele inferior leads evolved	hypo rca, nLvf	1	30	1.1	12.6	13000	3.1	39
48	1	0	7	0	0	0	0	0	0	0	0	0	1	1	1	1	1	1		4	0	114	130	80	2	1	1	186	6	st ele anterior leads	hypo lad,mild Lvd	1	38	1.5	12.6	14200	2.68	36
41	1	0	7	0	0	0	0	0	0	0	0	0	0	1	1	0	1	0	family h/o cat	5	0	46	160	102	1	1	1	121	5.3	st ele inferior leads evolved	hypo rca, n Lvf	1	19	1.1	17.9	16000	3.12	51.7
66	1	0	7	0	0	0	0	0	0	0	1	0	1	1	0	0	1	0		4	0	96	130	70	1	1	1	138	5.2	st dep inferior leads	hypo rca, n Lvf	1	32	1.4	9.9	8300	3.4	29.6
62	1	1	9	14	1	1	0	0	0	0	0	0	0	0	1	0	1	1		0	6	60	130	80	2	2	1	369	6.2	st dep anterior leads	hypo Lad, mod Lvd	1	43	1	10	9400	1.8	31
54	1	1	7	1	2	0	0	0	0	0	0	0	1	0	1	0	0	0	family h/o cat	20	0	92	160	100	1	1	1	264	9.6	st dep anteroseptal leads	hypo Lad, mod Lvd	1	30	1.2	13.3	9800	2.84	40.2
68	2	0	7	0	0	0	0	0	0	0	0	0	1	1	0	0	0	0	post menopause	4	0	80	160	110	1	1	1	172	5	st ele lateral leads	hypo Lad, mod Lvd	0	41	1.1	13.3	8100	2.34	40.7
54	1	0	7	0	0	0	0	0	0	0	0	0	0	0	1	1	1	0		2	0	86	130	90	1	1	1	156	6	st ele anterior leads	hypo Lad, n Lvf	1	24	1.4	16.5	11500	3.74	48.5
56	1	1	7	2	1	1	0	0	0	0	0	0	0	1	0	1	0	0		3	0	90	120	80	1	1	1	120	6.7	st ele inferior leads evolved	hypo rca mild Lvd	1	19	1.4	16	13500	2.36	48
50	1	1	7	5	1	1	0	0	0	0	0	0	1	1	0	0	1	0		1	0	86	140	96	1	1	1	233	8.3	st ele anterior leads	hypo rca mild Lvd	0	16	0.9	14.8	17600	2.6	42.9
53	1	1	7	1	1	2	0	0	0	0	0	0	1	1	1	0	1	0		5	0	70	120	80	1	1	1	144	5.9	st dep anterior leads	no rwma, n Lvd	0	31	1.3	17.7	12000	4.2	45
55	1	1	7	1	1	4	0	0	0	0	0	0	0	0	0	0	0	0		4	0	50	100	70	1	1	1	96	5	st ele lateral leads evolved	hypo Lad, mild mod Lvd	0	43	1	15.3	9200	4	39
65	1	0	7	0	0	0	0	0	0	0	0	0	1	1	0	0	0	0		22	0	80	150	100	1	1	1	198	6.9	st dep anterior leads	hypo Lad, mild Lvd	1	19	1	12.2	14300	1.97	37.9
35	1	0	7	0	0	0	0	0	0	0	0	0	0	1	1	1	1	0		4	0	72	120	90	2	1	1	155	6	st ele anterior leads	hypo Lad, mild Lvd	0	16	1	16.8	5700	3	49
50	1	0	7	0	0	0	0	0	0	0	0	0	0	1	0	0	0	0		4	0	86	120	70	2	2	1	107	5.3	st dep inferior leads	no rwma, n Lvf	0	32	1.2	13.3	6200	3.2	36
74	1	0	7	0	0	0	0	0	0	0	0	0	1	1	1	1	1	1		10	0	84	110	70	1	1	1	145	5	st ele anterior leads	hypo rca, mild Lvd	0	46	1.3	11.5	8600	2.98	34.8
70	1	1	7	10	1	1	0	0	0	0	0	0	1	1	0	0	0	1		3	0	60	140	80	1	1	1	148	8.7	st depinferolateral leads	hypo rca, Lcx, mod Lvd	1	35	1.2	12.4	9500	4	35.58
47	1	1	7	5	2	1	0	0	0	0	0	0	0	1	1	0	0	0		20	0	60	110	70	1	1	1	105	10.2	st dep anterior leads	no rwma, n Lvf	0	33	1.1	14.9	16100	2	42.2
26	1	0	7	0	0	0	0	0	0	0	0	0	0	1	1	1	1	0		6	0	98	110	70	1	1	1	79	4.9	st ele anterior leads	hypo Lad mod Lvd	0	30	1	14.5	13000	3.6	42.9
40	1	0	7	0	0	0	0	0	0	0	0	0	1	1	1	1	1	1		6	0	118	110	90	2	2	1	182	9.2	st ele inferior leads evolved	global Lv hypo, severe Lvd	1	29	1.3	13.7	13000	1.87	39.5
70	1	0	7	0	0	0	0	0	0	0	0	0	1	0	0	0	0	0	hypothyroids	1	0	40	96	70	1	1	1	167	5.2	st ele inferior leads	hypo rca, n Lvf	0	20	1.2	12.6	12300	3.43	37
70	2	1	7	4	1	1	0	0	0	0	0	0	1	1	0	1	0	0	post menopause	0	6	102	180	120	2	2	1	182	6.4	st ele inferior leads evolved	hypo rca,n mild Lvd, mod mr	0	31	1.4	13	17100	2.15	38
60	1	0	8	0	0	0	0	0	0	0	0	0	0	1	0	0	1	0		10	0	92	140	90	2	2	1	299	7.1	st ele lateral leads	hypo Lad,severe LVD	1	25	1.4	14.8	17100	3.05	45.8
74	1	1	10	20	1	1	0	0	0	0	0	0	1	1	0	0	0	0		2	0	86	50	0	1	1	1	406	9.3	st ele inferior leads	hypo rca, n lvf	0	26	1.2	11.4	7200	2.39	34.5
56	1	1	7	5	1	1	0	0	0	0	0	0	1	1	0	1	1	0		7	0	84	110	70	1	1	1	122	7.6	st ele anterior leads evolved	hypo rca, n Lvd	0	15	0.9	13.8	8500	2.27	40.3
64	1	1	8	6	1	1	0	0	0	0	0	0	1	1	1	1	1	1	family h/o cad	22	0	108	140	80	1	1	1	327	7.2	st eleiferior leads evolved	hypo rca, n Lvd	1	17	1.4	10.2	12500	4	32.6
54	1	0	7	0	0	0	0	0	0	0	0	0	1	1	0	1	0	0		2	0	80	130	80	1	2	1	185	5.2	st ele anteroseptal leads evolved	hypo Lad mild Lvd	1	30	1.4	14.4	10100	4.2	43
55	1	0	7	0	0	0	0	0	0	0	0	0	1	1	1	0	1	0		6	0	82	150	90	1	1	1	214	6	st dep anterior leads	hypo rca, mild Lvd	1	23	1	16.4	18800	4	43
62	1	0	7	0	0	0	0	0	0	0	0	0	1	1	0	0	1			0.5	0	69	140	80	2	1	1	120	6.1	st ele anteroseptal leads evolved	hypo lad, n Lvf	0	48	1.2	9.5	9000	3	31.7
50	1	1	5	3	1	1	0	0	0	0	0	0	0	1	0	0	0	0		23	0	100	110	60	2	1	1	180	6.3	st ele anterior leads evolved	apical vsr,hypo Lad severe LVD	0	17	1.2	13.5	12300	2.7	40.2	8
72	2	1	8	10	1	1	0	0	0	0	0	1	1	1	0	1	0	0	post menopause	6	0	124	140	80	1	2	1	292	9	LBBB	hypo Lad, mild Lvd	1	29	1.5	8.5	6300	2.9	25.5
56	1	0	77	0	0	0	0	0	0	0	0	0	1	1	0	0	1	0		4.5	0	94	130	80	1	1	1	130	5	st ele anteior leads evolved	hypo Lad, mod - severe LVD	0	19	1.1	14.5	14000	3.2	44.4
55	2	0	7	0	0	0	0	0	0	0	0	0	1	0	0	0	0	0	Post menopause	24	0	82	130	80	1	1	1	82	5	stdep anterior leads	hypo lad, n lvf	1	15	0.9	11.5	13500	3.67	33.7
64	1	1	7	1	1	1	0	0	0	0	0	0	1	0	0	0	1	1		22	0	66	110	50	1	1	1	109	6.5	st dep inferior leads	no rwma , n lvf	1	34	1.4	11	8300	3.89	32.9
63	1	0	8	0	0	0	0	0	0	0	0	0	0	1	0	0	0	1		3	0	86	140	90	1	1	1	110	5.9	st ele anterior leads	hypo lad servere LVD	0	17	1.3	15	12400	2.45	45
60	2	1	7	12	1	1	0	0	0	0	0	0	1	1	0	0	0	0		4.5	0	80	150	100	1	1	1	338	8.6	st dep inferior leads	no rwma, n lvf	1	31	1.2	12.8	13400	3.57	38.8

72	1	0	7	0	0	0	0	0	0	0	0	0	0	1	1	0	1	0	18	0	65	140	90	1	1	1	132	6	st ele inferior leads	hypo rca, mod lvd	0	40	1.4	13.8	7600	1.96	41.7
55	1	1	7	3	1	1	0	0	0	0	0	0	0	1	1	0	0	0	3	0	80	130	80	1	1	1	222	7.4	st ele anterior leads	hypo lad, n lvd	0	18	1.2	14	11600	2.49	42.3
42	2	0	7	0	0	0	0	0	0	0	0	0	0	0	0	1	0	0	20	0	86	130	80	1	1	1	86	4.7	st dep inferior leads	no rema, n lvf	0	19	0.9	13.2	7700	3.3	39.5
39	1	0	7	0	0	0	0	0	0	0	0	0	0	1	1	1	1	1	22	0	76	120	80	1	1	1	273	11	st dep anterior leads	hypo lad, n lvf	0	18	1.2	17	11200	2.9	45
63	1	1	7	20	1	3	1	0	0	0	0	0	1	1	0	1	0	0	18	0	80	150	100	1	1	1	198	7	st ele inferior leads	hypo rca, n lvf	1	33	1.3	12.7	10500	2.4	38
50	1	0	7	0	0	0	0	0	0	0	0	0	0	1	1	0	0	0	4	0	80	120	80	1	2	1	80	5	st ele inferiorleads evolved	hypo rca, n lvf	0	23	1	12.5	9900	3.2	38.2
40	2	0	7	0	0	0	0	0	0	0	0	0	1	1	0	0	0	0	2.5	0	72	130	90	1	1	1	113	4.8	st ele anterior leads evolved	hypo rca, mod lvd	0	27	1.4	11.2	9800	1.9	33.4
60	1	0	7	0	0	0	0	0	0	0	0	0	0	1	0	0	1	0	4	0	54	130	90	1	1	1	201	9.2	st ele inferior leads evolved	hypo rca, mild lvd	0	24	1.2	15.7	4000	2	45.4
71	2	0	7	0	0	0	0	0	0	0	0	0	1	1	0	0	0	0	6	0	94	170	90	1	1	1	66	6	st dep lateral leads	hypo rca, n lvf	1	19	0.8	11.4	9500	1.9	34.8
60	1	1	7	6	1	1	0	0	0	0	0	0	1	1	0	0	0	0	12	0	70	170	80	1	1	1	165	6	st ele inferior leads evolved	hypo rca, n lvf	1	40	1.5	12.7	10000	2	40
57	1	0	7	0	0	0	0	0	0	0	0	0	1	0	0	0	0	0	10	0	86	140	90	1	1	1	89	5.9	st dep lateral leads	no rema, n lvf	0	30	1	13.3	6800	2.38	41
45	1	1	7		1	1	0	0	0	0	0	0	1	1	0	0	1	0	4	0	72	160	90	1	1	1	125	7.9	st dep inferolateral leads	hypo rca, n lvf	1	15	0.8	12.9	12900	3	39
38	1	0	7	0	0	0	0	0	0	0	0	0	0	1	0	0	1	0	1	0	112	160	120	1	2	1	172	6	st ele anterior leads	hypo Lad, mild lvd	0	25	1.2	16.1	15600	3	49
60	1	0	7	0	0	0	0	0	0	0	0	0	0	1	0	0	0	0	24	0	96	110	90	1	1	1	180	6.4	st ele inferior leads evolved	hypo rca , mild lvd	1	21	0.9	11.6	5800	3.5	36.4
45	2	0	7	0	0	0	0	0	0	0	0	0	0	1	1	0	1	0	2	0	56	100	80	1	1	1	264	7.4	st ele inferior leads	hypo rca, n lvd	0	28	1.2	15.2	12300	3.7	46
55	1	0	7	0	0	0	0	0	0	0	0	0	1	0	0	0	0	0	20	0	86	140	80	1	1	1	93	5	st dep anterior leads	no rema, n lvf	0	32	1.1	11.5	7700	1.75	33.5
60	2	0	8	0	0	0	0	0	0	0	0	0	1	1	0	0	0	0	3	0	45	70	50	2	1	1	151	6	st ele inferior leads 2 blovk	hypo lad, sever LVD	0	33	1	9.9	12600	2.56	31
56	2	1	7	2	1	1	0	0	0	0	0	0	1	1	1	0	1	0	23	0	86	200	120	1	1	1	320	7.1	st dep anterior leads	no rewma, n lvf	0	26	1.3	13.5	5500	1.85	40
60	1	0	7	0	0	0	0	0	0	0	0	0	1	0	0	0	0	0	21	0	100	130	90	1	2	1	152	5.6	st ele anterior leads evolved	hypo lad, mod lvd	0	35	1.3	10.5	12900	3.76	30.6
44	2	0	7	0	0	0	0	0	0	0	0	0	1	1	1	0	0	0	12	0	90	110	90	1	1	1	123	6	st dep anterior leads	no rwma , n lvf	1	21	1.2	15.9	13900	3	46.3
53	1	0	3	0	0	0	0	0	0	0	0	0	1	0	0	0	0	0	2	0	70	120	100	1	1	1	113	5	st ele anterior leads	hypo lad, mod lvd	0	42	1.3	12.8	15300	3.5	39.1
54	1	0	9	0	0	0	0	0	0	0	0	0	0	1	1	0	1	0	1	0	74	110	80	1	1	1	126	6	st ele inferior leads	hypo rca n lvf	0	23	1	14	10400	3.5	42
72	1	1	7	25	1	3	0	0	0	0	0	0	1	1	0	0	0	0	4	0	120	150	100	1	2	1	400	7.3	LBBB	hypo lad sever LVD	0	48	1	11.6	16500	4	30.1
57	1	1	9	4	1	1	0	0	0	0	0	0	1	1	1	0	1	0	1	0	70	150	90	1	1	1	140	5.5	st dep anterior leads	hypo lad, n lvf	1	15	1.1	14.6	10200	2	41
72	1	0	8	0	0	0	0	0	0	0	0	0	1	1	0		1	0	4	0	78	170	110	2	1	1	396	6.4	st ele inferior leads evolved	hypo rca n lvf	1	17	1.4	14.4	16500	3.06	41
50	1	1	9	2	1	1	0	0	0	0	0	0	1	1	1	0	0	0	3	0	76	120	90	1	1	1	252	7.9	st dep anterior leads	hypo rca n lvf	1	21	1.3	13.5	14400	3.3	43
65	1	1	7	25	1	2	0	0	0	0	0	0	1	1	0	0	0	0	0	0	130	160	110	2	2	1	437	10	st dep anterior leads	hypo lad sever LVD	1	34	1.2	11.8	11400	3.7	38.7
64	2	0	7	0	1	0	0	0	0	0	0	0	0	0	0	1	0	1	1	0	86	70	0	1	1	1	247	5.8	st ele anterior leads	hypo lad mild lvd	1	31	1.4	11.7	15000	2.9	28.8
57	1	1	8	5	1	1	0	0	0	0	0	0	1	1	0	0	0	0	0	0	66	120	70	1	1	1	197	6.2	st dep lateral leads	no rwma n lvf	0	35	1.1	12.5	7400	2.6	37.8
56	2	1	7	4	0	1	0	0	0	0	0	0	1	1	0	0	0	0	1	0	104	130	90	1	2	1	384	7.7	st ele anterior leads	hypo lad sever LVD	0	44	1.3	13.5	8900	3.24	42.4
53	2	1	7	1	1	1	0	0	0	0	0	0	0	1	0	0	0	0	5	0	94	90	70	1	1	1	128	7.8	st ele anterior leads	hypo lad mild lvd	0	27	1	12	10500	2.56	30
59	1	1	7	2	1	1	0	0	0	0	0	0	0	0	0	0	0	0	2	0	96	110	90	2	1	1	210	8.4	st ele anterior leads	hypo lad mild lvd	0	17	0.9	13.3	8300	2.95	39.9
63	1	0	8	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	8	0	42	110	90	2	2	1	350	5.6	st ele anterior leads evolved	hypo lad, mod lvd	0	48	1.4	13.98	11800	4	40.5
50	1	1	7	2	1	1	0	0	0	0	0	0	1	1	1	1	1	0	6	0	72	130	94	1	1	1	100	5.6	st dep inferior leads	hypo rca n lvf	1	45	1.2	17.2	14300	3.44	45.5
46	1	1	7	7	1	1	0	0	0	0	0	0	0	1	1	1	1	1	3.5	0	96	100	70	1	1	1	185	7.6	st ele anterior leads evolved	hypo Lad, mild lvd	0	36	0.6	12.7	12100	2.2	36
70	1	1	8	6	1	1	0	0	0	0	0	0	1	1	0	0	0	0	4.5	0	88	140	90	1	1	1	307	6.6	st dep inferior leads	hypo rca, mild lvd	1	22	0.7	11.8	12000	2.75	39.2
50	2	1	13	6	1	1	0	0	0	0	0	0	1	1	1	0	0	0	1	0	94	130	90	1	2	1	592	7.8	st ele inferior leads	hypo rca, mild lvd	0	29	1	11.5	13300	3.863	35.8
69	1	1	7	20	1	2	0	0	0	0	0	0	1	1	0	0	0	0	14	0	88	140	90	1	1	1	178	6.8	st ele inferior leads evolved	hypo rca, n lvf	1	18	0.6	13.1	9000	2.8	43.8
65	1	1	7	10	1	1	0	0	0	0	0	0	1	1	0	1	0	0	5	0	114	150	110	1	2	1	378	9	st dep anterior leads	hypo lad, n lvf	0	49	1	11	16000	3.5	35
53	2	1	8	4	1	1	0	0	0	0	0	0	0	1	0	0	0	0	3	0	94	120	90	2	1	1	328	9	st ele inferior leads	hypo rca, mild lvd	0	25	1.4	13.7	9300	3	41.5
63	1	1	8	15	1	2	0	0	0	0	0	0	1	1	0	0	0	0	20	0	102	140	96	1	1	1	325	7.5	st dep lateral leads	hypo rca, mild lvd	0	43	1.4	13.5	4600	1.9	39.4
74	2	0	7	0	0	0	0	0	0	0	0	0	1	1	1	1	0	0	2	0	98	150	100	1	2	1	216	6.1	st ele inferolateral leads evilved	no rwma, n lvf	1	32	1.5	13.5	4800	2.1	41.7
62	1	1	7	10	1	1	0	0	0	0	0	0	1	1	1	0	1	0	24	0	98	180	100	1	1	1	213	7.4	st dep anterior leads	hypo Lad, mild lvd	1	20	0.8	14.1	4400	2	42.1
42	1	0	7	0	0	0	0	0	0	0	0	0	0	1	1	1	1	1	6	0	96	146	100	2	1	1	254	7	st dep lateral leads	no rwma, n lvf	1	13	1.1	16.4	8400	4	46
65	1	0	7	0	0	0	0	0	0	0	0	0	1	1	0	0	1	0	5	0	112	200	120	2	2	1	180	8.4	st dep anterior leads	hypo lad, n lvf	1	32	1.3	12.3	7000	3	38.5
62	2	1	13	10	1	1	1	0	0	0	0	0	1	0	0	0	0	0	1	0	150	220	110	2	2	1	537	7.7	st dep anterior leads	hypo lad, n lvf	1	28	0.8	13.8	12400	2.88	44.2
47	1	1	7	10	1	1	0	0	0	0	0	0	1	1	0	0	1	0	21	0	86	140	80	1	1	1	195	6.1	st dep inferior leads	no rwma, n lvf	0	21	1.3	11.2	9500	3.89	35

Abbreviations used in master chart

DM +/- 1= diabetes persent, 0= absent

Sex : 1 = male, 2 =female

Duration of hospital in days

Duration of diabetes mellitus in years

Treatment history 1=Oral hypoglycemic agents, 2=lnsulin, 3=Both

Target organ damage

Retinopathy 1 =present, 0=absent

Nephropathyl =present,0=absent

Previous cardiac events 1 =present,0= absent

Cerebrovascular events 1 =present, 0= absent

Prior diabetic complications 1=present, 0= absent

Risk factors

HTN-hypertension, 1=present, 0=absent

Dyslipidemia,1=present,0=absent

Smoking, 1 =present,0=absent

Alcoholism, 1=present,0=absent

Stress, 1=present,0=absent

Obesity, 1=present,0=absent

Chest pain and dyspnea in hours

Heart rate in rates/minute

Blood pressure in mm/hg

Cardiovascular system: normal=1, 2=abnormal

Respiratory system examination 1=normal, 2=abnormal

Abdominal examination 1= normal,2=abnormal

Investigations:

Hb-Hemoglobin: gm%

HCT- Haematocrit: %

Total count and platelet count: cells/mm3

Bt-bleeding time and ct- clotting time in minutes

RBS- Random blood sugar: mg%

HBA1C: in%

Urea and creatinine: mg/dl

Cardiac enzymes (CPK) positive =1, negative=0

Adverse cardiac events:

0 = normal

1 =left ventricular failure

2=arrhythmias

3=cardiogenic shock

Condition at discharge:

Recovered satisfactorily =1; residual dysfunction =2 ;death =3

Diagnosis:

STEMI=1,NSTEMI=2, unstable angina=3

KEY:

- DM – Diabetes Mellitus
- dur of hos – duration of hospital stay
- dur of DM – duration of Diabetes Mellitus
- type – treatment received for diabetes(OHA,insulin or both)
- retinop – retinopathy
- PVD – peripheral vascular disease
- pre car e – previous cardiac events
- nephro – nephropathy
- CVA – cerebrovascular accidents
- DM comp – diabetic complications
- HT – hypertension
- dyslip – dyslipidemia
- alsoho – alcoholic
- cp hrs – chest pain in hours
- HR – heart rate
- BP SYS – systolic blood pressure
- BP DIA – diastolic blood pressure

- CVS – cardiovascular system
- RS – respiratory system
- PA – per abdomen
- card enzy – cardiac enzymes
- S Creat – serum creatinine
- PCV – packed cell volume
- CT – clotting time, BT – bleeding time
- ADV Car Events – adverse cardiac events
- cond @ dis – condition at discharge

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This is to certify that the project work titled "A PROSPECTIVE STUDY ON THE CORRELATION BETWEEN THE ADMISSION DAY GLYCEMIC STATUS AND IN- HOSPITAL OUTCOME OF ACUTE CORONARY SYNDROME IN DIABETIC AND NON DIABETIC PATIENTS"
proposed by DR.JYOTHIPRIYA JYOTHINDRAKUMAR of K.A.P.V. Govt.Medical college, Trichy as part of fulfillment of M.D course in the subject of GENERAL MEDICINE during the academic period of 2010-2013 by The Tamilnadu Dr.MGR medical university has been cleared by the ethical committee.

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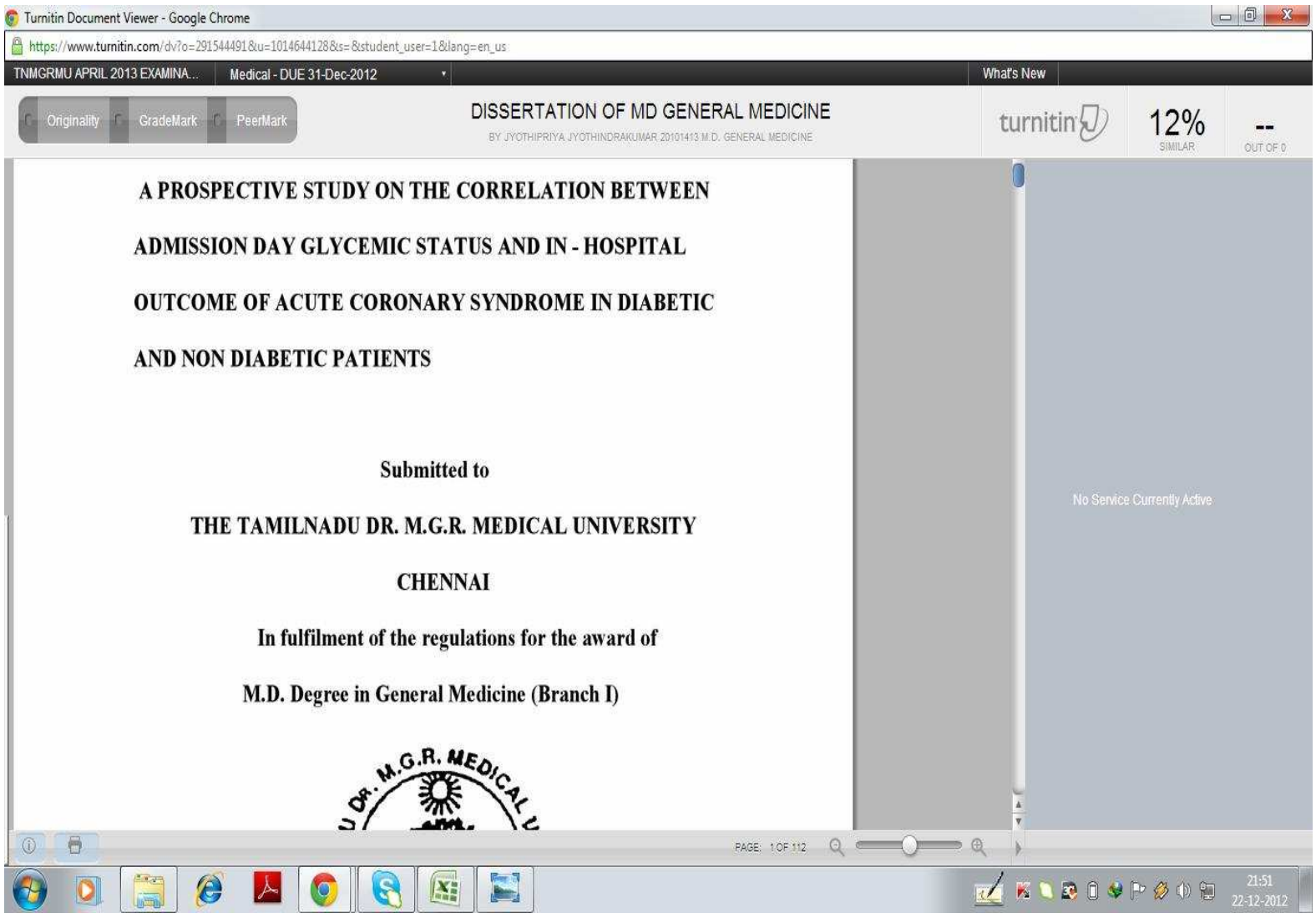
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Submitted to
THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY
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In fulfilment of the regulations for the award of
M.D. Degree in General Medicine (Branch I)
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